IN THE UNITED STATES DISTRICT COURT

FOR THE EASTERN DISTRICT OF PENNSYLVANIA

IN RE: ASBESTOS PRODUCTS LIABILITY

LITIGATION (NO. VI)

CIVIL ACTION NO. MDL 875

CIVIL ACTION NO. 5-92-88

----------x

This Document Relates To:

CONWED CORPORATION,
Plaintiff,

v.

UNION CARBIDE CHEMICALS AND PLASTICS COMPANY, INC.,
Defendant and Third-Party Plaintiff,

v.

OWENS-CORNING FIBERGLAS CORPORATION, ET AL.,
Third-Party Defendants.

----------x

KELLEY, DRYE & WARREN, L.L.P.
101 Park Avenue
New York, New York 10178

July 7, 1999
10:35 a.m.

DEPOSITION OF DR. EDWARD ILGREN

PRIORITY-ONE COURT REPORTING SERVICES, INC.
899 Manor Road
Staten Island, New York 10314
(718) 983-1234

A P P E A R A N C E S:

STICH, ANGELL, KREIDLER,
BROWNSON & BALLOU, P.A.
The Crossings, Suite 120
250 Second Avenue South
Minneapolis, Minnesota 55401
BY: ROBERT D. BROWNSON, ESQ.
Attorneys for the Plaintiff,
Conwed Corporation

RUDNICK & WOLFE, ESQS.
203 N. Lasalle Street, Suite 1800
Chicago, Illinois 60601
BY: MICHAEL R. GOLDMAN, ESQ.
Attorneys for the Plaintiff,
Conwed Corporation

FOLEY & LARDNER, ESQS.
Firstar Center
777 East Wisconsin Avenue
Milwaukee, Wisconsin 53202-5367
BY: TREVOR J. WILL, ESQ.
Attorneys for the Defendant and
Third-Party Plaintiff, Union Carbide
Chemicals and Plastics Company, Inc.

KELLEY, DRYE & WARREN, L.L.P.
101 Park Avenue
New York, New York 10178
BY: ALAN J. GERSON, ESQ.
Attorneys for the Defendant and
Third-Party Plaintiff, Union Carbide
Chemicals and Plastics Company, Inc.

I N D E X

E X H I B I T S

REQUESTS

PAGE NO.	
1025	Insert number of anomalous rat?

DR. EDWARD ILGREN,

called as a witness, having been first duly resworn according to law, testifies as follows:

DIRECT EXAMINATION BY MR. BROWNSON:

Q Good morning, Dr. Ilgren. We are continuing here. Let me ask you, since last we spoke, have you been out to Kings City or the Coalinga deposit or Calidria mine?

A No.

Q Have you had an opportunity to see or review any new information, since the time of our last deposition, from the mine or the area out there?

A Say that again. New information from where?

Q From out of the Calidria mine or mill or the deposit out there. I'm thinking in terms of any medical records, any reports, any air sampling, anything like that?

A No.

Q Have you read any information about any cases of mesothelioma in the Kings City area?

A No.

Q Have you examined or worked on any other cases for Union Carbide, other than this particular case involving Calidria exposure, since we last spoke?

A Yes.

Q Do you know where that exposure arose from. Was it a plant?

A Allegedly the ore bodies.

Q Was this any kind of an injury case or did it involve a worker of some sort?

A A policeman.

Q And this was a policeman where, in Kings City?

A Highway patrol.

Q Now, I had heard something about a case involving a policeman, and this must be the one; but I confess to you, I don't have any particulars.

Is this a highway patrolman who alleges he somehow got sick from working in the area of the Coalinga ore deposit?

A You are asking me what he alleged?

Q Well, what he is contending.

A As I recall, he alleges to have gotten his mesothelioma from Coalinga fibers that drifted twenty miles through the air into his highway patrol

car as he drove up and down Route 5 between 1978 and 1983 and also from two days of pig hunting on the ore body.

Q Do you know, has this highway patrolman filed a lawsuit?

A Yes.

Q Do you know what his name is?

A Waggoner.

Q Do you know who his lawyer is?

A Wartnick, Chaber.

Q Do you know how his name is spelled?

A I think it's W-a-g-g-o-n-e-r.

Q Is Union Carbide a defendant in that suit or is someone else the defendant; do you know?

A Union Carbide was a defendant and settled out.

Q Are there any remaining defendants, as far as you know?

A There is one but I don't recall what the name is.

Q Do you know if KCAC was a defendant?

A No, I don't know.

Q This highway patrolman Waggoner, is he dead or alive, as far as you know?

A He died in January.

Q January of 1999?

A Yes.

Q Do you know if this was a pleural mesothelioma or a peritoneal?

MR. WILL: Objection. I object to the form. He doesn't necessarily know it's a mesothelioma.

Q Did Mr. Waggoner contend it was a pleural or a peritoneal?

A I can't recall at the moment.

Q Do you know if any of his tissue was preserved for examination?

A I don't know.

Q Are you still involved in the case or did you cease your involvement?

A I'm still involved.

Q But you don't recall who this remaining defendant in the case is?

A No.

Q Are you still involved for Union Carbide or for somebody else?

A Somebody else.

Q You just can't recall who that is?

A I don't recall the name of the defendant.

Q Do you know, are there any other exposures being alleged in this case, other than this

Calidria exposure?
A Yes.
Q Is this other defendant, whose name you can't recall, are they in it as a result of some other exposure?
A Not to my knowledge.
Q How do they come in it in terms of the Calidria exposure; do you know?
A I don't recall.
MR. WILL: By Calidria, are you referring to the entire ore deposit?
MR. BROWNSON: That's a good point.
Q I should say Coalinga exposure.
A That's right.
Q Do you understand this other defendant to be somehow a defendant related to the Coalinga ore deposit, but it's not Union Carbide and it's not the Calidria mine itself?
A I don't know if it's the deposit or some other aspect. I don't know precisely how the exposure arises. Still, it's Coalinga.
Q Do you know any other scientists or physicians who are working on that case, either on behalf of Mr. Waggoner or on behalf of Union Carbide or anyone else?

A In this case, the wrongful death case?
Q Yes, the Waggoner case.
A No.
Q Is there anyone who worked on it at any time that you know that is no longer involved?
A Yes.
Q Who is that?
A Mark Shanker, Davis and Bill Nickelson.
Q Now, you said Mark Shanker?
A Yes.
Q And then you said Davis. You mean from the University of Davis?
A Right.
Q And Nickelson?
A Right.
Q What had Nickelson done on that case?
A I don't remember.
Q Do you know, was he attempting to contend that there was some sort of fiber drift, as he likes to call it?
A I think so, yes.
Q Was he attempting to make any calculations that you are aware of?
A I can't remember his disclosure. I read it. I can't remember the details.

Q As far as you know, was there an expert disclosure prepared by the lawyers that you read or did you see any actual reports or depositions or anything like that?
A From them?
Q From anybody else.
A Yes.
Q Yes?
A Yes.
Q What was it?
A An expert's disclosure.
Q So what you saw was an expert's disclosure that the Wartnick firm prepared from Nickelson?
A Yes, and Shanker.
Q You didn't see any of their actual work?
A No, I don't think so.
Q Are you aware of any pathologist who has examined the case at any time for anybody?
A That case?
Q Yes.
A No.
Q Is Dr. Chatfield involved in that case at all?
A At the present time?

Q Yes.
A No.
Q Do you know if he ever was?
A I think so, but I can't recall.
Q Do you know if he will be in the future?
MR. WILL: How can he know that?
MR. BROWNSON: He might have heard that they were going to get some tissue and send it to Dr. Chatfield.
Q I'm just asking if you know?
A I don't know.
Q Are you aware of any plans that Dr. Chatfield will be involved in the case? That's all I'm asking.
A No.
Q Now, let's turn to something a little closer to your heart, which is your series of papers in "Indoor Built Environment."

First of all, can you tell us anything more about paper numbers 4 and/or 5? How are those coming along?

Will those be published or when? Do you have any information on that?

MR. WILL: Which one of those questions are you asking him to answer?

MR. BROWNSON: I can break it down.

Q Where does paper number 4 stand?

A Well, we hope to have it finished by the end of the summer and the same with paper 5.

Q Is there any research ongoing at the present time that you are doing in connection with papers 4 or 5 or is your work done and you are now just writing?

A My work is done. I'm writing.

Q How about Dr. Chatfield, id he still doing any work; do you know?

A Not to my knowledge.

Q Have you made any additional examination of the archived material from Drs. McConnell and Pinkerton that you had looked at before in connection with papers 4 or 5?

A No.

Q Do you know when papers 4 and 5 might be published?

A No.

Q Do you intend to publish them or have them published in "Indoor Built Environment" or some other --

A Yes.

Q In "Indoor Built Environment"?

A Yes.

Q Now, I would like to turn your attention to paper number 3 or installment number 3 of your paper, which is entitled, "Lack of Biopersistence."

Do you have a copy there?

A I don't have one with me.

MR. BROWNSON: Do you have one for him, Trevor?

MR. WILL: Actually, no.

MR. BROWNSON: Let's mark it as Exhibit 39.

MR. WILL: It's already been marked.

MR. BROWNSON: Do you know what number it was?

MR. WILL: Well, it's 38.

MR. BROWNSON: We will call it Exhibit 38.

MR. WILL: Document entitled, "Coalinga Fibre: A Short, Amphibole-Free Chrysotile" was marked Defendant's Exhibit 38 for identification last time, so the record is clear.

Q You now have in front of you, Dr. Ilgren, Exhibit number 38, which is the paper subtitled, "Lack of Biopersistence," correct?

A Correct.

Q Authored by you and Dr. Eric Chatfield?

A Yes.

Q Now, I will try to move through this in fairly short order; but let's turn first to the materials and methods section, which is the second page of the paper on page 99.

Do you see that section?

A Yes.

Q First of all, are you the author of this section?

A I can't remember. I wrote a substantial part; but I can't remember what Eric -- Eric might have added something to it.

Q Do you know what he -- if he added to it, do you know what that might be?

A I can't remember. We traded drafts.

Q As you sit here today, do you know anything that specifically could have been authored by Dr. Chatfield?

A No.

Q Now, with respect to the materials and methods, as I understand that section in a scientific paper, that's the section that informs the reader, up front or towards the start of the paper, the nuts and

bolts of the materials and methods, if you will; is that correct?

A Yes.

Q Let me ask you this. Can you describe for us specifically what you actually did; and by you I mean you personally, in terms of the materials and methods for paper number 3?

A Specifically with regards to what?

Q Well, anything that is described in the materials and methods section. I'm trying to understand the work that you did.

A Can you ask me specifically what -- focus on something and say that I do this, because I'm confused.

Q Did you examine any tissue in connection with paper number 3?

A No.

Q Did you do any microscopic analysis?

A No.

Q Do you know if Dr. Chatfield did?

A I believe not.

Q Again, if we go to the materials and methods section it states: "The methods used to perform elemental analysis and the measurement of asbestos fibre volume and density are as described by

Pinkerton et al. [10]."

Do you see that?

A Yes.

Q Now, does that sentence describe some work that you did or some work that Pinkerton did?

A At the end there I state, "All of the morphometrical analyses were conducted by Professor Kent Pinkerton."

Q So to speed this along, would it be fair to say that all of the morphometrical analyses, which is described in the materials section, was work done by Pinkerton and not by you?

A Right.

MR. GERSON: We have to go off the record for a moment.

MR. BROWNSON: Sure.

(A discussion is held off the record.)

Q The morphometrical analyses conducted by Professor Kent Pinkerton, as described in the materials and methods section of your paper, are historical work by Pinkerton, aren't they?

A Historical?

Q In other words, they were done fifteen, twenty years ago?

A Yes, that's true.

Q They were not any new work done in connection with this particular paper?
A Not, not to my knowledge, though, I don't recall. He might have done something in 1994 or 1995; but I don't recall. I don't know exactly when he stopped doing these analyses; but they certainly weren't done in the past couple years.
Q And they were not done in connection with this particular paper, were they?
A Could you rephrase that?
Q Yes. Dr. Pinkerton's morphometrical analyses, as the ones described in the materials and methods section, were not done for purposes of this paper, were they?
A Well, I don't know. He asked me to help him write up the work. So to that extent, I guess -- I mean, they were aimed at forming a publication, which presumably would have taken this form.
Q Well, when you say he asked you to write up the work, he never came to you and asked you to publish data on the work he had done back in the late '70s and early '80s, did he?
A No. In 1991, when we corresponded, he sent me his data and said I'm free to write these up in whatever form I want. So that's what I meant.

Q And then when you undertook that task to write these series of papers, you described the materials and methods we see here on page 99, correct?

A Right.

Q But you describe them as materials and methods that had been done by Professor Pinkerton at some point in the past?

A Right.

Q And the topic of paper number 3 is biopersistence, right?

A Right.

Q If we could generalize the theme of paper number 3, would it be to go back to these old rat studies that Dr. Pinkerton had done while a graduate student and try to discern from these data the biopersistence of these three kinds of asbestos in the lungs of those rats?

A With the exemption that, I think, he did some work subsequent to being a graduate student, that would be correct.

Q As I read through paper number 3, you argue that the three different types of chrysotile asbestos have different biopersistence in the lungs of these rats in Dr. Pinkerton's study, correct?

A Right.

Q And you base that upon these various measurements of silica found in the lung tissue of those rats, correct?

A Partly.

Q Well, is there any -- was there any measurement of actual asbestos fibers from the lungs of those rats?

A Sure.

Q And where do we find that in your paper?

A Fiber volume and density. There are three indices. There is the silica ashings, that's the morphometric fiber volume density; and then there is the stuff that I noticed on the presence and absence of dust-laden macrophages, which is on page 102 column 2 paragraph 4.

Q Did anyone do any microscopic analysis of asbestos fibers in the lungs of these rats?

A Yes.

Q Where do we find that in your paper?

A Well, it's table 2 and 3 and the column page section I just told you about on page 102.

Q Where do I see in table 2 or 3 analysis of asbestos fibers?

A All fibers. One is a fiber volume, the volume

of the tissue component under consideration occupied by fiber and fiber density is the so-called density or number of fibers present in, again, certain tissue component as listed.

Q Let me ask this question. Did Dr. Pinkerton actually count fibers in this tissue or did he measure silica?

A No, in tables 2 or 3 they would be "fiber counts."

Q So as you understand, Dr. Pinkerton actually counted asbestos fibers in the tissue in table 2 and 3?

A Yes, per materials and methods citation, "The methods used to perform elemental analysis and the measurement of asbestos fibre volume and density are as described by Pinkerton et al. [10]," which is his 1984 paper.

So the details of the manner in which the fibers were counted were in the 1984 paper.

MR. BROWNSON: Let's mark this as Exhibit 39.

(A copy of "The American Journal of Pathology" is marked as Plaintiff's Exhibit 39 for identification, as of this date.)

Q I will show you, doctor, what we marked

as Exhibit 39, which is a paper of "The American Journal of Pathology" by Dr. Pinkerton and some other doctors entitled "Fiber Localization and Its Relationship to Lung Reaction in Rats After Chronic Inhalation of Chrysotile Asbestos."

My first question will be, is that paper the reference 10 that is cited in your paper number 3, Exhibit 38?

A Yes.

Q So if I understand what you have told us, the analysis of actual asbestos fibers as opposed to measurements of silica that Dr. Pinkerton made in the lungs of rats comes out of this paper, Exhibit 39?

A Yes.

Q Is there anything in that paper, which is your reference, in Exhibit 39, which was published in 1984 by Dr. Pinkerton, that talks about the biopersistence of the three types of chrysotile asbestos to which these rats were exposed?

A I don't recall. I think it may allude to the persistence of the Jeffrey fiber. I don't believe it mentions UICC/B and Coalinga.

Q Now, going to your paper, Exhibit 39, let's start then with the --

MR. GERSON: Exhibit 39?

MR. BROWNSON: 38, sorry.

Q Let's start with the next heading after materials and methods, which is entitled results.

Do you see that?

A Right.

Q Page 99?

A Yes.

Q Again, to try to summarize this, what you have done, you have drawn some conclusions about the biopersistence of these three different types of asbestos by looking at the silica content determination in the lungs of these rats and the specific fiber density and volume changes within different compartments of the lungs of the rats; is that right?

A And the presence and/or absence of dust-related macrophages, but that's correct.

Q Let's start with the silica content determinations of the lungs. First of all, did you do any actual analytical work with respect to the silica content determinations of the lungs of these rats?

A No.

Q Again, this was work that Dr. Pinkerton had done back in prior years?

A Yes, plus -- there were other data that fall into this table that may or may not have been Pinkerton's that I found at the NTP archive, namely, the Jeffrey fiber one-day exposure twelve-month follow-up. That's on page 100 top of column 2.

Q And you found that in the archive, but you are not sure who actually did it?

A Yes, it was with the other raw data; but I'm not sure whether -- Pinkerton did not send me the raw data. The raw data, as enumerated in the table, came out of the archive.

Q So this is some material that you found in the archive; but you are not quite sure who actually did it in the first place?

A That's right.

Q Now, with respect to the silica lung content determination in these rats, as I understand it, you looked at these various results that Dr. Pinkerton had gotten back when he made the rats inhale the three types of asbestos in the late '70s and early '80s, correct?

A Right.

Q Again, you didn't actually look at any rat lung tissue, you looked at the numbers generated by Dr. Pinkerton when he did that work?

A Or whoever, but that's right.

Q With the exception of this one thing you just told us about, it was work by Dr. Pinkerton?

A To my knowledge, that's correct.

Q And then you basically published that data from Dr. Pinkerton and/or this other unknown person in paper number 3; and then you drew some conclusions from it, correct?

A Right.

Q Again, focusing on the lung silica content portion of this data, as I understand, what that shows is how much silica was in the lungs of these rats at different points of time, right?

A Right.

Q Silica is a component, if you will, of chrysotile asbestos, right?

A Correct.

Q So at least with respect to the silica lung content portion of your analysis, you are not actually describing asbestos fibers per se, what you are describing is the amount of silica which presumably could have come from the asbestos fibers?

A Presumably, right.

Q Do you know, in fact, that all of the silica in the lungs of these rats did come from the

asbestos fibers or could some of it have come from other places?

A It could have come from other places. I don't know what they would have been. There is obviously some in the control annals which weren't exposed. So there is clearly some kicking around the lungs which isn't asbestos derived.

Q Would you agree with me that a more accurate way to determine how much asbestos is in the lungs of rats would be to analyze the number of asbestos fibers in those lungs as opposed to measuring silica content?

A Yes, we state that in the paper. I mean, this has just been the traditional methods used by Vogner and Davis and others in the past who also conclude or point out, as you do, that it would be, perhaps, more accurate, if you are interested in fiber localization and identification, to look at the individual fibers.

Q As I understand, there is nothing we can determine from looking at silica content with respect to fiber length, is there?

A No.

Q To do that, we actually have to look at fibers, measure their lengths and count them and that sort of thing?

A Right.

Q Now, is there anything in paper number 3, the biopersistence paper, which describes the size distribution of the asbestos fibers which were found in the lungs of these rats exposed by Dr. Pinkerton?

A Not to my knowledge.

Q If you had the opportunity to actually design a study where you were going to make rats inhale asbestos and then determine biopersistence, would you agree that it would be interesting to determine the size distribution of the fiber in the asbestos the rats were breathing and how the different sizes of fibers stayed or were cleared from the lungs over time?

MR. GERSON: What is your question?

MR. BROWNSON: Read it back.

(The record is read back.)

A He did it all in the '86 paper for Jeffrey, but I don't know -- I couldn't find it for Coalinga or UICC/B.

Q Just so the record is clear, by Jeffrey, you mean the Jeffrey mine chrysotile asbestos?

A Right.

Q Which is one of the three types of asbestos that Dr. Pinkerton made the rats breathe?

A Right.

Q So in a paper published by Dr. Pinkerton in 1986, he shows how the different sizes or lengths of the Jeffrey chrysotile asbestos persisted or was cleared over time from the lungs of rats; but you couldn't find that for the other two types, the UICC/B chrysotile or the Coalinga chrysotile?

A Right. I'm referring to my reference 9.

Q So would you agree with me that, in your paper number 3, you then took Dr. Pinkerton's data and tried to make some effort to determine how all three types of asbestos persisted in the lungs of the rats over time?

A Right.

Q And you had to do that yourself, because Dr. Pinkerton did not -- had not done that, he had just done it with one of the three, the Jeffrey, right?

A Maybe I don't quite understand your question.

MR. GERSON: I don't either.

Q Is there any published data anywhere, other than what you show in your paper number 3, that shows the biopersistence of the Coalinga asbestos that these rats in Dr. Pinkerton's study breathed?

A No.

Q Is there any published data, other than what you show in your paper number 3, that shows that the biopersistence of the UICC/B asbestos that Dr. Pinkerton's rats breathed?

A Not to my knowledge.

Q Again, to try to summarize, if we look at the state of the published data from this rat inhalation study that Dr. Pinkerton did, before you published your paper number 3, we can find information about the biopersistence of the Jeffrey asbestos but not the other two?

A No, I don't agree with that.

Q Where would we find the other two?

A In terms of -- well, your question was about biopersistence.

Q Right.

A Can you read that question back?

(The record is read back.)

Q Now, I was excluding this paper. Maybe the question was not clear. Let me ask a different question.

Before you wrote your paper number 3 that we are looking at, before then, was there any published data showing the biopersistence of the other two kinds of asbestos in the lungs of these

rats?

MR. GERSON: Published data?

MR. BROWNSON: Right.

A The other two, either Coalinga and UICC/B?

Q Right.

A Yes.

Q Where would we find that?

A For Coalinga, it's the Muhle data and for UICC/B it would be references in table 5 as they are expressed in silica content, ashing and there may be some other studies of UICC/B that I haven't come across.

Q Well, there may be but --

A I'm not aware of.

Q So in terms of published data concerning the biopersistence of Coalinga asbestos, as I understand it, this Dr. Muhle published about that and you talk about that in your paper, correct?

A Right.

Q Because he also did a rat inhalation experiment over in Germany, right?

A Right.

Q Again, I'm trying to get my arms around the published data, if you will, showing biopersistence of Coalinga asbestos in the lungs of

rats.
A Right.
Q You have attempted to make some calculations of your own, which we will get to in a moment, in your paper number 3; and Dr. Muhle had published some results from his rat inhalation study?
A Right.
Q Is there any others?
A To my knowledge, no.
Q Then with respect to UICC/B chrysotile asbestos, which is another of the three types that Dr. Pinkerton exposed his rats to, you reference some studies of silica lung content in your table number 5 that other people had done, right?
A Right.
Q We will get to that in a minute; but does that then constitute the published data concerning biopersistence of Coalinga asbestos or UICC/B asbestos in the lungs of rats that you are aware of?
A Yes -- wait. I reference another study by Bellmann and I could never get those data. It's referred to on page 105 column 2 paragraph 1 in the -- starting the tenth line. That was an intratracheal injection study, I believe.
Q So page 105 of your paper, column 2, the

first paragraph, you make a reference to a study by Bellmann, at which is your reference number 23, and this involved UICC/B chrysotile not Coalinga, right?

A No, I believe he looked at both. I would have to get the paper out and look at that again, the Bellmann paper; but that was an intratracheal study.

Q That was an injection study where they injected asbestos into the rats? The rats didn't breathe it?

A No.

Q I don't want to get into this issue again about injection versus inhalation studies. We talked about that before.

A We did.

Q You are not a big proponent of injection studies, I take it. You prefer the inhalation studies?

A Well, to some extent it's all related; but I obviously like to look at the inhalation studies.

Q Let me go back and try to summarize. If we can get around the published data concerning inhalation studies of Coalinga asbestos and UICC/B chrysotile asbestos, those would be the ones that we talked about a minute ago?

A Right.

Q Which would be Dr. Pinkerton's work and Dr. Mulhe's work, right?

A As I recall.

Q And also those things you talked about in your table 5 that we will get to?

A Right.

Q Now, table 1 of your paper, which is at page 100, let's go to that. I hope to be brief here.

Would it be fair to say that in table 1 you are attempting to present some of Dr. Pinkerton's data about the lung silica changes over time with respect to the three types of asbestos?

A That's correct.

Q And you also presented his data concerning the control rats, too, correct?

A That's right.

Q Now, if we look at the data of Dr. Pinkerton's that you presented in table 1 with respect to the Coalinga, or for that matter, with respect to either of the other two fiber types, I don't see any data concerning the lung silica content twenty-four months.

A They didn't do it.

Q With respect to the silica lung content, are you aware of any data that exists where that

could be examined in the lungs of those now long dead rats at twenty-four months?

A For Pinkerton's study?

Q Right, or is that just something that is not available?

A It could be buried in the archives.

Q But you didn't find it, right?

Well, let me ask a different question. You didn't present it in table 1, we know that. Does that indicate you didn't find it?

A Are you asking me whether I looked for twenty-four month data and I didn't find it or I made an attempt to get tissues to do a twenty-four month digestion to fill in the blank?

What are you asking?

Q Well, actually both are good questions. Let's start with the easy part first.

We know that these rats, the lungs of these rats, were examined at twenty-four months, because, for example, we see that in table 2 with respect to the fiber volume changes, right?

A Right.

MR. WILL: Some rats were examined.

Q The lungs of some rats were examined twenty-four months after they first began exposure to

the asbestos, right?

A Right.

Q But with respect to the lung and silica content, you don't present any data on that in your paper, right?

A Right. Pinkerton sent me, in 1991, a histogram; and the histogram only had three and twelve-month data; and I asked Kent did he have any twenty-four month data and he said no. I didn't ask him why.

I might have, but I don't recall why there were no twenty-four month data sent to me; but there were no twenty-four month data sent to me. So I went back, and when I was in the archive, I found the raw data and this is the presentation of the raw data, which underlie the data that he sent me in histogram form.

Q But even looking at that, you were unable to present in this paper any twenty-four month data concerning lung silica content?

A I didn't find it. I looked for it.

Q Now, would you agree with me that going back these twenty years when this Pinkerton study was designed, this was not designed to be a biopersistent study?

A No, that's not true at all. No, he had -- I mean, case in point, per the 1984 paper, fiber localization, and the 1986 paper, which is -- per his 1986 paper, they were looking at the manner in which the fiber density and the fiber volume change over time.

In Pinkerton's thesis, he talks, as I recall, about the accumulation, the clearance, the retention of three fiber types and the differences between them. I mean, I think it was part and parcel of the whole aim of the effort to look at persistence and clearance.

Q Well, if that was part and parcel of the whole aim and effort, would you agree with me that there was no paper actually published by the studies' designers which set forth the biopersistence of the three types of asbestos in the lungs of these rats?

A Well, he sent me two manuscripts, which they sent to some journal. I don't remember the name. It was initially rejected, the data in that.

So there is nothing published, to my knowledge, of fiber volume density change data in a presently scientific peer reviewed paper, aside from this one; but I wouldn't say that it wasn't their aim from the outset to present data, not only on fibrosis

and tumors, but also on this particular aspect of the work. So I wouldn't agree with that.

Q Do you know for a fact that there was an intent of the study, back when it was originally designed, to measure biopersistence?

A Well, that presumes knowledge that I wouldn't necessarily have, because I wasn't there in the beginning of the study; but on the basis of the data Kent sent me and the manuscripts he sent me and the raw data, it would seem to be that that was part of the aim of the work.

Q So you are drawing that conclusion, correct?

A Right.

Q Now, if we look at your table 1, which is the silica content changes in the rat and lungs over time, what we see, if we focus on the Coalinga, what we see is that the overall average goes from 610 micrograms per lung after three months of breathing the Coalinga to 574 after twelve months, correct?

A Correct.

Q I don't have my calculator with me; but would you agree that with respect to the biopersistence of the Coalinga, it decreases between three and twelve months about ten percent or less?

A I don't think you can say that. I think you can just look at this and say, there is some trends here. You get the first -- I mean, trying to perhaps move this along, I mean, you can say that you have got clear deposition of fiber for all three fiber types in the three months.

Secondly, at twelve months, there is clearly much more Jeffrey deposited than the other two. Beyond that, on the basis of the data that's presented, I don't think you can say much. At the twelve-month follow-up, with the one day exposure, you get clearance; and that's something that I put in as a kind of control; but beyond -- I mean, beyond that, what I have just said, I don't think you can say too much more.

Q Well, the fact of the matter is, from the lung silica content data presented in your table 1, we don't see much clearance of the asbestos from the lungs of these rats by up to twelve months, do we?

A No, but if you look -- the text of the paper directs the reader's attention to table 5; and the discussion text also indicates that these are -- I don't know if I would use the words crude measurements, but crude measurements. If you look at table 5 --

Q I will get to table 5 in a minute and I don't mean to cut you off.

MR. WILL: You are.

Q I'm trying to limit my question to table 1. I'm trying to do this table by table.

With respect to silica, table 1, which is silica content over time, you will agree with me that that data does not show much clearance up to twelve months of the asbestos types, does it?

A I don't think you can tell. I mean, it could be an equilibrium, as Pinkerton suggests. So there could be significant clearance and significant deposition.

I think with respect to Jeffrey, we are comparing three to twelve months. You can have a deposition with much lower clearance; and I think that's what you see when you look at the 750 circa at three months and then you look at twelve months and you get a thousand.

Q Let me ask you, with respect to the Coalinga asbestos, your table 1 indicates that after twelve months the rats had almost as much Coalinga asbestos in their lungs, as measured by lung silica content, as they did at three months, correct?

A Right.

MR. WILL: Well, I object to the question. It measures silica micrograms per lung. As you pointed out before, that is not an exact measurement of asbestos fibers.

MR. BROWNSON: I was attempting to say that.

Q Table 1, presumably, is presented in your paper to show something, correct?

A Right.

Q It does not support your hypothesis that most of the Coalinga is cleared at least by -- over time, does it?

A Well, there is a twelve-month time point. You start the inhalation exposure at zero and you keep it on to twelve months; and I believe what I said in the paper, which is what basically Pinkerton says in his papers and thesis, is that from the zero to three to twelve months, during which these animals were being exposed to very high doses of Coalinga, a steady state has been established between the deposition of the lungs and the clearance of the lungs. So the clearance is very efficient.

If you look at the Jeffrey, and again, Pinkerton points this out, it has a much higher concentration of fiber and you have a significant

increase between 734 and 753 at three months up to a thousand at twelve months; and all that is simply reflected in the other data. It's consistent with what others have seen, such as Muhle, and it's consistent with the fiber volume changes in table 2.

Q These rats were exposed to asbestos for twelve months, correct?

A Right.

Q Then they were allowed to wander about in their cages for another twelve months; and at twenty-four months, their lungs were getting examined, right?

A Right.

Q But we don't have that particular data with respect to the lung content of silica expressed in table 1. We will get to that in other tables, right?

A Right.

Q So at least during the time they were breathing the asbestos up to twelve months, what we see is that they breathed, presumably, asbestos into their lungs by three months and it pretty much stayed that level through twelve months for the Coalinga and the UICC/B and it increased by about twenty-five percent for the Jeffrey; is that fair to say?

A Right.

Q We glean that from table 1?

A Right.

Q What we can't glean from table 1 is what happened over the next twelve months when they weren't breathing asbestos. We have to look at other things to find that, correct?

A Right, and I point that out in the paper.

MR. GERSON: This may be a good time to take a five-minute break.

(A short recess is taken.)

Q With respect to the lung silica content, do you know what the percentage of silica is in the Coalinga fiber?

A No.

Q Is it different than in the Canadian chrysotile?

A I don't know.

Q So I take it that you don't know what the percentage of the silica is in the UICC/B or Jeffrey Canadian fibers?

A No. These are in the standard tables; but I don't have the numbers offhand.

You mean, the part per million per silica?

Q Whatever percentage or part silica is of

the asbestos.

A I don't know.

Q So now the second thing you do after examining the lung silica content changes over time in table 1 is you then move to table 2 and examine the fiber volume changes over time, correct?

A Right.

Q Now, in reviewing table 2, I don't see reference to the control rats. Is that in here somewhere?

A There were no control data.

Q For the lung volume changes?

A Right, nor for the density.

Q Which was table 3?

A Right.

Q Now, are there any other published asbestos biopersistence studies that use fiber volume changes, such as we see in table 2, that you can refer us to?

A Well, there is Pinkerton '84, '86, and I think in Pinkerton '90, they certainly do that. I think there are some papers by Chang and Crapo, his group; and there may be other people who have also done other determinations of fiber volume and density, which I haven't read.

Q With respect to those Pinkerton papers, that's this same study you are talking about in table 2, right?

A Yes, though Crapo may not be.

Q But do you know what it is that Crapo did or didn't examine concerning fiber volume changes?

A His specific study?

Q Right.

A No, I don't know.

MR. BROWNSON: Off the record.

(A discussion is held off the record.)

MR. BROWNSON: Why don't we mark as Exhibit 40 this document. It is a copy of Lung Reaction to Chronic Inhalation of Three Types of Chrysotile Asbestos During the Lifespan of the Fischer 344 Rat, University Microfilms International, copyright 1982 by Pinkerton, Kent Ed.

(A copy of Dr. Pinkerton's 1982 thesis is marked as Plaintiff's Exhibit 40 for identification, as of this date.)

Q Dr. Ilgren, we marked as Exhibit 40 a copy of the thesis by Dr. Kent Pinkerton for his Ph. D. in 1982; and this is something that you have read, I take it?

A Yes.

Q At page 103 of the thesis, Dr. Pinkerton writes: "Compared to the control value, the interstitial matrix volume was 60 percent greater in male rats exposed to Coalinga chrysotile, 54 percent greater in males exposed to UICC/B chrysotile and 143 percent greater in male rats exposed to Jeffrey chrysotile."

He is talking now about twelve months. Do you recall seeing those data?

A Yes. You said 143 percent.

Q Yes.

A Yes.

Q Then he continues at page 103 and writes: "The total volume of the interstitial matrix continued to increase during the postexposure period in control and treatment groups of both sexes."

Do you recall that reference?

A I believe so.

Q Do you want to see it?

A No, that's okay.

Q What I'm kind of leading into is the lung volume measurements which you --

A That doesn't talk about fiber. It's just the matrix. Can you read it again? I don't think --

MR. WILL: Why don't you show it to the witness?

A It's the interstitial matrix volume. That's not fiber volume.

Q What is that?

A That's the matrix within the interstitial, which is collagen or precollagen. That's not fiber.

Q What causes the matrix in the interstitial?

A There is an aging effect, because you see it in the controls and it could also be and/or due to fiber and/or due to non-specific things. The animals get viruses -- you want me to show you the section?

Q You are correct. It's interstitial matrix volume. So that's different than fiber volume.

A Right. We address IMV in paper number 1.

Q Now, at page 104 of your paper, now, I'm in the discussion section.

A Right.

Q At this point, what you have done is you have set out in your tables the silica lung content data from Dr. Pinkerton and the lung volume data from Dr. Pinkerton that we have just looked at; and you are now discussing what that means, correct?

A I guess so, right.

Q One thing you say on page 104, and I'm looking under the discussion section in the second column about a third of the way down.

You describe how the three-month and twelve-month lung data of these rats exposed to Coalinga and UICC/B asbestos are consistent with, and I'm quoting from you, "were consistent with other studies of these two same fibre types that made silica content measurements in investigations conducted under nearly identical exposure intensity and duration conditions"; right?

A Right.

Q So what you are doing here, I take it, is finding support or corroboration from these other studies with respect to the Coalinga and the UICC/B, right?

A Right.

Q And we talked earlier in the deposition that these are other published data concerning biopersistence of the Coalinga and the UICC/B asbestos, right?

A Right.

Q Now, with respect to the other published data concerning biopersistence of Coalinga, you told

us earlier that you found that in this rat inhalation study by Dr. Muhle from Germany, right?

A Right.

Q And that's what you are now talking about in page 104 of your paper we are looking at, right?

A Right.

Q And I will show you a photocopy of a paper, which I believe is the paper by Dr. Muhle that you are talking about.

Let me show it to you and I will ask you, is this the Dr. Muhle page that shows the Coalinga data that you reference in your paper as reference number 15?

A Yes.

MR. BROWNSON: Why don't we mark that as 41.

(A copy of "The Annals of Occupational Hygiene" is marked as Plaintiff's Exhibit 41 for identification, as of this date.)

Q I will show you, Dr. Ilgren, what's been marked as Exhibit 41. Just for the record, is this the copy of the paper by Dr. Muhle referenced by number 15 in your paper?

A Yes.

Q To try to move this along, this paper is

entitled, "Inhalation and Injection Experiments in Rats to Test the Carcinogenicity of MMMF."

That means man-made mineral fibers, correct?

A Yes.

Q And they are examining whether some certain man-made mineral fibers can cause cancer in rats; and they are comparing them, if you will, to crocidolite asbestos from South Africa and chrysotile asbestos, which is the Calidria asbestos, right?

A Right.

Q Now, in your paper at the bottom of the second column of page 104 you state that: "The short fibre Coalinga sample used by Muhle et al. were cleared more than any other type of chrysotile and to the same extent (about 90 percent)."

Did I read that right?

A "And the short fibre Coalinga sample used by Muhle et al. were cleared more than any other type of chrysotile and to the same extent (about 90 percent)."

Q Is that right?

A Yes.

Q And when you say "any other type of chrysotile," I didn't see any other type of

chrysotile in Muhle's study.

Is there another type of chrysotile?

A I think I was referring to my table 5. I have to read it again.

Q And your table 5 references some other studies?

A Yes, a whole bunch of other studies.

Q So when you say it was cleared more than any other type of chrysotile, you mean as compared to these other types in the other studies in table 5?

A Yes.

Q Let's go back and look at Muhle's study where you say the Calidria was cleared to about 90 percent. I want to look at that, okay?

A Okay.

Q If you would turn to page 759 of Muhle's paper at table 4. This table is entitled "Fibers (SEM) Retained in Lungs at Various Times From the Start of the Inhalation Study (a) Number of Fibers Per Lung, all sizes, and in Parenthesis, Fibers Longer than Five Microns."

A Okay.

Q I'm now looking at table 4 and they are showing the clearance of four different things -- what table 4 shows is how much stuff is retained in

the lungs of the rats at different periods of time, right?

A Right.

Q And they are talking about four different things. It's two man-made mineral fibers, which are the glass fibers; and then the chrysotile asbestos and then the crocidolite, right?

A Right.

Q Now, if you look at this, I'm focusing now on the chrysotile, which is the Calidria. It shows that at six months these rats retained 398, which I assume, means 398 million fibers or does it mean something else?

A No, that's what it says, if you look at the top header.

Q So with respect to the Calidria chrysotile, these rats that breathed in Dr. Muhle's study after six months retained 398 million fibers in their lungs and at twelve months retained 347 million and at twenty-four months retained 223 million fibers in their lungs?

A Of all sizes.

Q I didn't bring my calculator along, but the difference between 398 million fibers at six months and 223 million at twenty-four months is about

forty percent, correct?

A But you didn't read my paper very carefully. We address the issue of why that's not really so. He analyzes the six and twelve months at SEM and twenty-four months at TEM. So he is finding probably ten to one hundred times more using a higher powered methodology at twenty-four months.

Plus, for all sizes, I don't really know what the lower end is; but I think if you actually look at the fibers greater than five at six, twelve and twenty-four months, it's going 39, 33 and then 11. If you adjust the 11 for the TEM, the first two are done by SEM, the third one is done by TEM, you are going to find ten to one hundred times more.

So, in fact, I would say you are probably going from 39 to 33 to one or less than one and that's a very substantial drop; and it comports well with table 5 data.

Q Now, you say and you talk about that in your paper, but I'm now examining table 4 of Dr. Muhle's paper and Dr. Muhle does not say that, does he?

He just presents these data. He didn't say that in this paper that the twenty-four month data ought to be reduced by one hundred times, does

he?

A That's just common knowledge.

Q Does he say that anywhere in his paper?

A He actually does talk about the unreliability of the measurements done with Calidria, I believe, with TEM. So he alludes to that. Hang on a second. I can find that.

Well, he is referring to injection study, but that was with TEM and he says that sizing of the fibers is very difficult resulting in numbers and fiber dimensions. They are not very reliable.

Q Well, table 4 in this study, that's his inhalation study where the rats are breathing, right?

A Right.

Q If we look at Dr. Muhle's published data presented in table 4, what we see is that, from six months to twelve months, there is less than a ten percent reduction in the chrysotile, correct?

Those were using the same technique, which is SEM, right?

A Right.

Q And then from six months to twenty-four months, there is about a forty percent reduction.

You say, no, it should really be more than that because he is using TEM?

A It's obvious.

Q That's what you say.

A No, that's what the literature would say. Dr. Langor would totally agree, if you are using SEM and then you go to TEM.

Q Dr. Muhle does not say that anywhere in this paper, does he?

A No, he doesn't actually explicitly say that in his paper.

Q Now, the only table that Dr. Muhle presents, as I read it, which actually says there is a significant reduction of the Calidria chrysotile is, in fact, table 5.

Would you agree with me in that?

A It may also be reflected in table 6 where Bellmann et al. '87 is cited.

Q But, again, I'm talking about Dr. Muhle's rat inhalation study.

A Okay, that's right.

Q Table 5, unlike the first two tables in table 4, rather than actual observed fibers, is a calculated fiber mass per lung, right?

A Right.

Q And Dr. Muhle calculates the fiber mass per lung using the technique in the King paper?

A Just say that again.

Q Dr. Muhle calculates the fiber mass per lung using the technique in King, by Dr. King, correct?

A King '55?

Q Right.

A I have to check. I thought Pinkerton was the one who -- I don't recall.

Q Well, if I represent to you --

A You want to show me where Muhle says how he analyzed, through what method he used, because I don't recall seeing that from Muhle.

Q Well, let me ask you a different question. Do you know by which method Dr. Muhle calculated these fiber mass per lung data in table 5?

A I have to read this for a second.

I think he just says, after low temperature ashing. He doesn't given any reference.

Q So do you know, then, how he calculated that or by what method he calculated it?

A If it's not cited, no.

Q Are you aware what you cite in your paper number 3, the studies by Dr. King in 1955?

A I think it's what Pinkerton used. That should be in materials and methods on page 99, column one

paragraph 1, "The silica content in lung tissue was determined after ashing by the method of King et al. 1955."

That's what Pinkerton used or whoever was working with Pinkerton.

Q So if I could summarize the findings by Dr. Muhle as published in his tables, which you say supports your contention that the Calidria fibers are not biopersistent, Dr. Muhle's data shows that when he measures the fibers, there is about -- there is a relatively small clearance; but when he calculates the fiber mass per lung, that's a larger clearance?

A That's not what I'm saying.

Q That's what I'm asking. That's what the table shows.

A Well, you can interpret the table for how you want.

Q I'm not asking to interpret it. I'm asking what does it show?

A Per the numbers in table 4, it shows for all fiber sizes a drop at twelve to twenty-four months from 347 to 223, which you gave me a percentage before.

Q I said forty but we can calculate it.

A And for all fiber sizes greater than five

microns, it goes from 33 to 11, which is what?

Q That's about a two-third reduction?

A So then there is almost a seventy percent reduction, even using those numbers for greater than five; but with table 5, if you look at the change in the fiber mass going from six to twelve to twenty-four months, you go from .31 to .29 to .03.

So that's the drop from .29 to .03.

Q That's a much larger drop?

A Right.

Q Again, that's the only one of the three tables using a calculation as opposed to an actual measurement of numbers, right?

A These are actual measurements. This is not a calculated -- they ashed the tissue. They measured the residue and this is what they got, as far as I understand.

Q It's described as a calculated fiber mass per lung, right?

A I know calculated is a funny word; but my understanding is, per the methodology, they ashed the tissues, they got the numbers and they are presenting for a certain number of animals. It looks like for three animals at each time point.

They have averaged them out and they got

0.31. As far as I can see, they are actual measurements.

Q In any event, though, you don't know what actual methodology was used for that calculation, right?

A No.

Q Now, would you agree with me, Dr. Ilgren, though, if you go back looking at table 4 at twenty-four months, using the TEM technique, which you have told us about, what Dr. Muhle found was that there were 223 million fibers in per lung of these rats of Calidria chrysotile, right?

A Right.

Q Regardless of what technique you use or how you measure it, 223 million fibers is a lot of fibers in a rat's lung, isn't it?

A Well, on the assumption that they are all -- you know, I don't even know if his cut-off is at the standard size, which would exclude things beneath that limit that weren't even considered to be fibers; but on the face of the statement 223 million "fibers," that would seem to be a lot of fibers.

Q He doesn't say "fibers." He just says fibers.

A Fine.

Q Do you know how much a rat's lung weighs?
A I can't remember.
Q Again, regardless of what technique he uses; and, in fact, in twenty-four months he uses TEM and in six and twelve he uses SEM, he finds Calidria asbestos longer than five microns in the lungs of these rats, doesn't he?
A Right.
Q So although you have consistently stated that Calidria is a short asbestos, you would agree that some portion of Calidria asbestos is greater than five microns in length and that some portion of Calidria asbestos greater than five microns in length got into the lungs of Dr. Muhle's rats and stayed in the lungs of Dr. Muhle's rats for twenty-four months?
MR. WILL: Objection. That's four questions. State them one at a time.
MR. BROWNSON: Well, let him answer that.
MR. WILL: Well, it's four questions.
MR. BROWNSON: You can answer.
MR. WILL: Read the question back.
(The record is read back.)
MR. WILL: As to my objection as multiple, it's also argumentative.

Doctor, feel free to ask him to rephrase it.

A I'm focusing on two -- the first part of your question says I have consistently said something; and the second part alludes to the presence of claimed fibers greater than five.

Not to be a major pain in the ass, but would you like to ask the first part of your question?

Q You would agree with me, Dr. Ilgren, that Calidria asbestos has fibers that are greater than five microns in length?

A Yes, there is a small percentage.

Q Do you know what percentage that is in a sample of Calidria?

A In water, it would be generally less than four or five percent.

Q Now, in Dr. Pinkerton's thesis at page 23 he describes the Coalinga asbestos, which is the same as the Calidria asbestos, that he looked at as having forty-eight percent fibers greater than five microns. You are familiar with that reference?

A Well, in error.

Q Well, it's in the air that the rats breathed, right?

A Well, that the fibers --

Q The rat breathes the asbestos out of the air and when that rat breathes it, according to Dr. Pinkerton, forty-eight percent is greater than five microns?

A Well, as an aerosol. He assumes he is measuring the fiber and his cut-off for short versus long, which is between thirty and forty microns, as indicated in his thesis.

Q Well, Dr. Pinkerton states that forty-eight percent of the Coalinga asbestos his rats breathed was greater than five microns in length, right?

A You are asking if it's correct that Dr. Pinkerton stated that?

The structures that he was measuring, yes, that's correct that he stated that.

Q Well, I can show you where he stated it.

MR. WILL: For the record, what is the reference?

MR. BROWNSON: The reference is at page 23 of the thesis.

MR. WILL: Which is Exhibit 40.

Q In fact, in the thesis, Dr. Pinkerton actually presents tables showing the size

distribution of the three asbestos, including the Coalinga, right?

A Right.

Q And you are familiar with the fact that some portion of the Coalinga chrysotile asbestos from the Union Carbide mine is greater than five microns in length?

A Yes.

Q If we then look at Dr. Muhle's rats that were made to breathe the Coalinga asbestos, their lungs contain some of those fibers that are greater than five microns in length, right?

A Again, I don't know if he is measuring fibers, pseudo-fibers, which are not really fibers. There is a possibility that there are some long fibers there.

Again, it's apples and oranges. He does six and twelve months with SEM and then he goes to twenty-four months and looks at the things using an instrument that is tremendously more powerful.

Q Let's just focus on this twenty-four month measurement, which is using this TEM.

Using that powerful transmission electron microscope, Dr. Muhle reports at page 759 in his paper, that you cite in your paper, that he is finding in lungs of his rats Coalinga asbestos

greater than five microns in length, right?

A Right.

Q And he doesn't say these might be pseudo-fibers. He reports them as fibers, right?

MR. GERSON: Well, hold on. I just want to make it clear for us, as well as the record. You are asking Dr. Ilgren to state what Dr. Pinkerton has stated?

MR. WILL: No, Dr. Muhle.

MR. GERSON: Dr. Muhle, rather. You are just asking him to affirm or deny that's what is in the report, right?

MR. BROWNSON: Right.

A Right.

Q In short, using the transmission electron microscope, Dr. Pinkerton reports that his rats that breathed Coalinga asbestos have Coalinga asbestos in their lungs greater than five microns, right?

A Because he couldn't see them with SEM.

Q Well, with SEM, he reports them too, doesn't he? Look at six and twelve months. He finds that using that technique as well, right?

A Well, that's when the animals are being continuously exposed to very high doses of Calidria.

Q And Dr. Muhle's rats, after six months of

exposure of Coalinga asbestos, using his SEM technique, had 39 million fibers greater than five microns in length in their lungs?

A Right.

Q After twelve months, they had 33 million Coalinga fibers greater than five microns in their lungs?

A Right, fibers, as he says, fibers.

Q And at twenty-four months, using a different technique, which is now the TEM, he measures 11 million Coalinga fibers greater than five microns in the lungs of those rats?

A Right.

MR. BROWNSON: Let's take a lunch break here.

(A lunch recess is taken at 12:35 p.m.)

(Afternoon session resumes at 1:45 p.m.)

Q If we turn our attention, Dr. Ilgren, to part 3 of your paper, the other study that I understand you relied upon to support your belief of the Coalinga studies that we see at table 5; and I'm now looking at table 5, which is on page 106 of your paper.

As I understand, from your text, you find support for the notion of clearance of Calidria in

the Muhle study, which we already talked about, and that's the first part of table 5; and then you also find support in the studies of the short fiber immediately beneath that; is that right?

A Yes.

Q Now, I'm looking at the category in table 5 which you have labeled as short, which is the second category there. Do you see that?

A You mean this?

Q Right.

A Again, from reading the text of your paper, I understand you to say that the studies of the short fiber will lend credence to the idea that the short fiber clears more rapidly.

Is that a correct statement?

A Where are you reading?

Q Well, I'm paraphrasing from the text. I can go to a reference, if you would like?

A Page 104, column two?

Q Exactly. So then, going to table 5 on page 106, I'm looking at your column headed clearance percentage. Do you see that one?

A Yes.

Q You cite Davis et al., which is a published paper in 1988, your reference 33, for a

clearance percentage of 89.2.

Do I have that right?

A Yes.

Q Now, in the Berman et al. paper, which is the Berman 1995 paper and your reference 29, you don't put a clearance percentage. Why is that?

A That was a study they did where they reanalyzed the dust clouds using TEM; and I think I put that in there just because it reassessed the same studies that Davis had originally done.

I think they gave some additional measurements for fibers per CC; but I don't think Berman has clearance. I have to go and double check.

Q But at least as far as your paper, you don't report a clearance percentage for that Berman cite?

A Right.

Q So in terms of actually getting a number, we need to look at Davis' study for a clearance percentage, right?

A Right.

MR. BROWNSON: Let's do that. Off the record.

(A discussion is held off the record.)

Q Your table 5 at page 106 of your paper

talks about a study of Davis et al. published 1988 in the "British Journal of Industrial Medicine." That is your reference 33; and I have just showed you the paper, which is your reference 33, and you are saying that's an error?

A Yes. The electrostatic charge pertains to this one at the bottom. You see normal charge, the very last one on that page. It says normal charge and draft 33. That's the one.

MR. BROWNSON: Okay, mark that paper.

MR. WILL: Is it supposed to be 28?

THE WITNESS: Yes.

MR. WILL: Reference 33 in the paper should be reference 28.

MR. BROWNSON: Let's mark that.

(A copy of "Effects of Electrostatic Charge on the Pathogenicity of Chrysotile Asbestos" is marked as Plaintiff's Exhibit 42 for identification, as of this date.)

Q I show you, doctor, what was marked as Exhibit 42, and this is a study by J.M.G. Davis and other authors and this is the reference described as reference 33 in your paper, right?

A Yes.

Q When you state at table 5 that reference

33 in the short fiber type category, you are not telling us that that's an error, there's really a different reference that supports that?

A Yes.

Q And the different reference that supports it is reference 28 in your paper?

A Yes.

MR. BROWNSON: Let's mark that as Exhibit 43.

(A copy of "Comparisons of the Pathogenicity of Long and Short Fibres of Chrysotile Asbestos in Rats" is marked as Plaintiff's Exhibit 43 for identification, as of this date.)

Q I show you what's been marked as Exhibit 43, and I will ask you if this is the paper which is, in fact, reference 28 in your paper?

A Yes.

Q Can you show us where in reference 28 of your paper, which is Exhibit 43, we find data that shows that 89.2 percent of the short asbestos was cleared in the lungs of rats?

A I think I just calculated that from the data. I don't think it's stated, though it might be stated. I don't remember.

Q Well, if it is stated, are you telling us that it would be found somewhere in that paper, Exhibit 43, your reference 28?

A Yes, you saw the lung burden data.

MR. WILL: Why don't you state that for the record.

THE WITNESS: It looks like 732 table 5.

Q So you are saying that the 89.2 clearance percentage that you report on table 5 of your paper for short fiber type is derived from table 5 at page 732 of Exhibit 43, which is your reference 28?

A I think so.

Q Now, one thing I do observe is that it appears as though the inhalation study of asbestos on rats referenced in both Exhibits 42 and 43, which are your references 33 and 28, involve the same group of forty-eight rats; is that right?

A No, I don't think so.

Q So these are different groups of forty-eight rats?

A Well, I think we are talking about -- you are saying my reference 28 and reference 33 --

Q Is that the same group of rats? That's all I'm asking.

A No, I don't think so.

Q So those are two different inhalation studies on two different groups of rats?
A Yes, short, long, discharged and non-discharged.
Q Were both of those studies using the same type of chrysotile asbestos?
A I don't remember. I think so, but I don't remember. I have to look. You want me to look?
Q Sure.
A Both studies meaning the two differences?
Q Right.
A I don't think so. The materials and methods in the reference 28 said that they used a grade 4T-30 sample, whereas in the other electrostatic charge paper they are using, I believe, a UICC/A.
Q Would you call that a short chrysotile, an intermediate or a long?
A Which?
Q The UICC/A.
A The UICC/A?
Q Right.
A I think the UICC/A is similar to UICC/B; but I'm not entirely sure. I think it's an intermediate to long.
Q Well, your reference number 33, which is

Exhibit 42, is a study by Dr. Davis and others where they are making rats inhale this UICC/A chrysotile asbestos, right?

A Right.

Q You don't know, as you sit here, whether that would be what you would call a short, intermediate or long fiber?

A I believe it's intermediate to long. It's not short. It's just a standard UICC/A sample.

Q Do you know how its size distribution compares to that of Calidria asbestos?

A I mean, there is more long fibers. I can't give you the exact percentages in each size category as I sit here.

Q And what type of chrysotile asbestos are the rats in Dr. Davis' study, which is your reference 28, Exhibit 43, exposed to?

A I think the 4T-30 is a derivation of Jeffrey, but I'm not sure.

Q Do you know if that is a short, intermediate or long?

A That's long.

Q Do you know how its size distribution compares to that of Calidria?

A I can't remember the percentile break down off

the top of my head. It's clearly termed in page 720. The long fiber dust cloud was generated directly from the 4T-30.

Q And just so we are clear now, going back to table 5 of your paper on page 106, the short, what you describe as short chrysotile study, is Berman et al., but that one doesn't have any clearance percentage data, correct?

A Right. I think I just put that in, the Berman et al. study, for the reader if they wanted to get more information about those particular Davis studies, because Berman et al. really looked at the Davis et al. studies; but you are right, it didn't have any clearance, as I recall.

Q So are you now telling us that the Davis study, which is your reference 33 and Exhibit 42, does not, in fact, involve short fiber?

A They didn't involve short fiber.

Q So when you place it in the short fiber category in your table 5, that's simply an error?

A No, reference 28 involves short -- the reference 28 is short versus long fiber and the second study I put down in table 5 is the fiber type that I label short; and that should refer not to Davis et al. 1988 (33), but it should refer to Davis

et al. 1988 (28).

Q So what you call Davis et al. 1988 (33), a short fiber type study, that is an error? It is not short?

A That's right.

MR. GERSON: But the error is the 33?

THE WITNESS: Right.

Q The error is you put the wrong study in the short fiber category, right?

A Right.

Q Just so I'm clear here, are you telling us that your reference 28, which is Exhibit 43, that's not short fiber either?

A Right.

Q So in order to fix your short fiber category on table 5, you can't just plug in your reference 28, Exhibit 43, because that's not short either?

A Sure you can. You just put a 28 here instead of a 33.

Q And if you do that, though, you just told us that was long to intermediate fiber in that study.

A No. We were talking about -- we are mixing, I think, exhibit numbers and reference numbers.

Q Then let's clearly go through them. We

have established that Exhibit 42, your reference 33, which your table 5 says is short, is not, in fact, short. We have got that much established, right?

A Right.

Q Now, I thought you were telling us that what should be in that short fiber category is your reference 28?

A Yes, which is Exhibit 43.

Q But I thought you told us that that study also was intermediate to long fiber?

A No, we were talking at that point, as far as I was concerned, about reference 33, Exhibit 42.

Q So if we looked, then, at your reference 28, which is Exhibit 43, that study involves some short chrysotile; is that correct?

A Yes, short compared to long.

Q You would put that --

A Well, that's just in the title.

Q If you were to correct your table 5, you would put that study in the short category, right?

A Yes, I would just put a 28 instead of a 33.

MR. GERSON: Does it still accurately describe Davis et al.?

THE WITNESS: Yes.

MR. WILL: Off the record.

(A discussion is held off the record.)

THE WITNESS: Actually, I misspoke. When you said is it still Davis et al., it's actually Davis and Jones.

Q You would agree with me, again, that your paper, part 3 of your paper, the purpose is to convince the reader that short, in general, and Calidria asbestos, in particular, is cleared from the lungs of rats more quickly than long?

A Reference 28.

Q Would you agree with me that your paper, part three of your paper, the purpose of part three of your paper is to convince the reader that short, in general, and Calidria asbestos, in particular, is cleared more quickly from the lungs of rats or more completely from the lungs of rats than long asbestos?

A Yes.

Q Would you agree with me that table 5 in your paper on page 106 is offered in support of that proposition?

A Yes, to some extent, yes.

Q Now, looking at Exhibit 43, which is your reference 28, this is the study by J.M.G. Davis and A.D. Jones where they made rats inhale both short fiber chrysotile and long fiber chrysotile?

A Right.

Q At table 5, which is that table you pointed to us a moment ago, they report on the clearance of both the short fiber and the long fiber, correct?

A Right.

Q Do you anywhere in your table 5 describe what they reported as the clearance of the long fiber chrysotile?

A It's on page 107.

Q Where do I find it on page 107?

A It's the third one down.

Q That's in the category entitled long?

A Yes.

Q And you report the clearance percentage as 54.1 percent?

A Yes.

Q And you report it as coming from that Davis et al. reference 33?

A Right.

Q But really it's the Davis and Jones reference 28?

A Right.

Q So that's just an error you made in your column of references there?

A Yes.

Q Now, even though this Berman et al. paper does not contain actual clearance percentages, you still cited it in your short category?

A I have to see it. Do you have a copy there? Do you have Berman's paper?

Q Yes, I do.

A Let me look at that, because I don't remember.

MR. BROWNSON: Off the record.

(A discussion is held off the record.)

Q I have showed you, Dr. Ilgren, my working copy of the Berman paper. Just for the record, is that the Berman paper?

A Yes.

MR. BROWNSON: Even though I have got some highlighting on it, why don't we mark it as an exhibit.

MR. GERSON: If you want, I can make a copy without the highlights.

MR. BROWNSON: Can we take a moment and do that?

MR. GERSON: Sure.

MR. BROWNSON: Let's do that.

(A short recess is taken.)

MR. BROWNSON: Mark that as Exhibit 44.

(A copy of "The Sizes, Shapes and Mineralogy of Asbestos Structures that Induce Lung Tumors of Mesothelioma in AF/HAN Rats Following Inhalation" is marked as Plaintiff's Exhibit 44 for identification, as of this date.)

Q Before we go to the Berman paper, Dr. Ilgren, I'm going to go back to this Davis and Jones paper, which is your reference 28, Exhibit 43, which is the one that should be in the short chrysotile category of your table 5, okay?

A Yes.

Q I was just noticing in that paper at page 730 there is a table 3 entitled, "Pulmonary Tumors and Mesotheliomas Found in Animals Treated with Long and Short Fibre Samples of Chrysotile Asbestos and in a Control Group of Rats."

Do you see that?

A I have a copy here. What's your question?

Q According to that table, it indicates that the forty rats that were exposed to long chrysotile had twenty-three tumors, forty rats exposed to short chrysotile had eight tumors and the forty-seven control rats not exposed to any asbestos had two tumors, right?

A Right.

Q So even though the short chrysotile had less tumors than the long chrysotile, it had four times as many as the control rats.

A Right. Is there a question?

Q No. Now, let's move to the --

MR. WILL: Just to be clear, that's not Coalinga chrysotile in that experiment, is it?

THE WITNESS: No.

Q Exhibit 44, this is the paper by Dr. D. Wayne Berman and other authors that you talk about at your table 5, right?

A Right.

Q I see one of those authors is Eric Chatfield. That is the same guy who is your co-author?

A Right.

Q And this paper has studies of the inhalation of nine different types of asbestos by a bunch of rats?

A The phraseology of the question, there were thirteen inhalation studies analyzed.

Q But different rats were exposed to nine different types of asbestos, right?

A Well, it looks like four chrysotile --

Q Well, I'm looking at the first line of the abstract where it says they were exposed to nine different types of asbestos dusts.

A Well, I don't know how he gets nine. If you look at table 1, there are four types.

MR. GERSON: What page is table 1?

THE WITNESS: 183.

Q If you look at the description, though, the chrysotile are broken down and so are the amosite and --

A Well, as far as I'm concerned, there are kind of sub-types and sub-variations to a type of asbestos. Not to be semantical, but in column one, table 1, fiber type, asbestos type. So anyway, it's semantical.

Q Are you saying this group of authors should not call this nine different types of asbestos, as they do in their abstract?

They should only call it three types with sub-groups?

A I would say they used four different types of asbestos of varying -- prepared in different ways. I would just have said it differently, that's all.

MR. GERSON: In fact, there are two columns.

Q Well, whatever the columns are, you agree with me in their abstract they call it nine different types of asbestos?

A Right, in their abstract.

Q And you don't agree with that terminology? Would you call it something else?

A I would have just worded the sentence differently, that's all.

Q If you look at their table 1, that's the table where they summarizes the data for animal inhalation experiments conducted by Davis and co-workers, right?

A Yes.

Q If you look at what they describe as the short chrysotile, that's that same short chrysotile that we were just looking at a moment ago in Dr. Davis' paper, which is Exhibit 43 and your reference 28; is that right?

A Right.

Q If you look at table 1 again under their category of short chrysotile, we see again, just like we saw in Exhibit 43, that that short chrysotile caused seven pulmonary tumors and one mesothelioma, correct?

A Right, being seriously contaminated with 1,170

long fibers per CC, which is discussed in Davis and Jones 1988 on page 733, I think, in adequate detail.

Q So you are saying that even though they call it short fiber, Davis calls it short fiber and you call it short fiber, it's seriously contaminated with long fiber?

A Well, that's what he says in his paper.

Q So it's really not short fiber?

A They made an attempt to make it short fiber, but they couldn't get out a lot of long fiber.

Q When you say a lot of long fiber, do you speak about fiber greater than five microns in length?

A Yes.

Q What percentage of this short fiber is greater than five microns in length?

A Say that again.

Q What percentage of that short fiber is greater than five microns in length?

A What they began with as the hand sample or in air.

Q What the rats inhaled?

A I don't know. I just know that as they indicate there. Despite their attempts to develop a method or to apply a method, that under other

circumstances were smaller amounts of fiber, despite that method to separate out long versus short, it just didn't work here.

So they had left over a large number of long fibers. Basically, he says, that while this certainly applies the samples of a few milligrams, it was not practical for the 1.5 kilograms needed for long term inhalation studies.

Q So the asbestos fiber Drs. Davis and Jones in Exhibit 43 described as short is, in fact, you are telling us, not all short. Some of it is long?

A Yes.

Q Nevertheless, Dr. D Wayne Berman, Kenny S. Crump, Eric J. Chatfield, John M.G. Davis and Alan D. Jones in their paper at page 183 table 1 call it short chrysotile, don't they?

A It's the same paper. They cross reference. In their table 1, they cross reference a reference, which is Davis and Jones 1988.

Berman et al. 1995 is merely a re-examination of all the Davis studies. So they are stating their reference 13 in Berman et al. 1995 is Davis and Jones 1988 comparisons of the pathogenicity of long and short fibers.

Q Let me ask it again. Drs. Berman, Crump, Chatfield, Davis and Jones, Davis and Jones being the original authors of Exhibit 43, and Chatfield being your co-author, in their paper, Exhibit 44, they don't make all these distinctions you do in table 1. They call it short chrysotile, correct?

A What do you mean?

Q What do they call that fiber in table 1 of their paper? Please read to me what they call it?

A Short, but they refer to reference 13, which is the paper in which the various distinctions are made by the same authors.

Q So a reader reading table 1 of their paper at page 183 would be told that that chrysotile is short; but if the reader bothered to go and read the reference 13, he might learn that some of that chrysotile was, in fact, long; right?

A No, you could see it in the fourth column, fifth column from left to right, it is fiber type, description, abbreviations, mass concentration and PCM. Under PCM is 1,170 fibers per milliliter there.

The reader, I think, would immediately realize that there was a significant number of long fibers in that short preparation, even if they didn't go back to reference 13 Davis and Jones of 1988.

Q Would that be obvious to the reader from looking at your table 5 as well?
A Yes.
Q Where does your table 5 show that those 1,170 fibers are greater than five microns in length?
A They are because they are PCM.
Q Where does it say that in your table 5?
A It says it in the footnote on page 107, two lines down.
Q So if a reader goes down to your second footnote, he would learn that some of the chrysotile fibers that you categorized as short are, in fact, long, correct?
A Right.
Q And he would also learn that some of the Coalinga fibers, which you were calling short in your paper, are also long?
A Right.
Q If a reader read the paper by Drs. Berman Crump, Chatfield, Davis and Jones, they might pick up that what those authors describe as short chrysotile, in fact, contains long chrysotile, even though it doesn't say that, because there is a reference that reads, PCM f/ml, right?
A Right.

Q And if a reader then went to Dr. Davis and Jones' paper, which is Exhibit 43, your reference 28, and read the paper closely, they would learn that what Dr. Davis and Jones called short chrysotile, in fact, contains some long chrysotile?

MR. GERSON: You said "some," right?

MR. BROWNSON: Right.

A Right.

Q And if a reader reading your part three paper went back to Dr. Pinkerton's original thesis, which describes the research upon which your part three paper is written, they would discover that some of that Calidria fiber you described as short is also long, wouldn't they?

A What would they be looking at to get that information?

Q Well, they could find that information from Dr. Pinkerton's thesis, couldn't they?

A Just sort of ask the question so it's kind of joined up into one question.

Q Well, they could find from Dr. Pinkerton's thesis that some of the Calidria asbestos you describe as short in your series of three papers is, in fact, long?

A And you would be citing what for that?

Q Dr. Pinkerton's thesis.
A What specific part of the thesis?
Q Please answer the question.
A I am. I'm just trying to -- you are referring to a thesis, which is a big piece of work; and you are saying somewhere in there he is saying something.
Can you just specify where in the thesis?
Q Okay, page 23 of the thesis.
A Okay, let's see page 23.
MR. WILL: It's a deposition exhibit.
MR. BROWNSON: It's Exhibit 40.
Q Dr. Pinkerton's thesis reads at page 23 and I quote --
MR. WILL: Why don't you show it to the witness?
Q "Combined fiber and fiber cluster length in Coalinga chrysotile shows a distribution with many fibers exceeding thirty microns in length."
Dr. Pinkerton reported that, correct?
A Yes, he reported that.
Q And his thesis at page 23 also shows that of the Coalinga asbestos that the rats breathed, forty-eight percent was greater than five microns in length, correct?
A Right.

Q Do your series of three papers anywhere make reference to the fact that some of the Calidria Coalinga asbestos is greater than thirty microns in length?

A That's part four. I don't recall -- I would have to go through the three papers. To my recollection, it does not mention that; but as we have discussed in the past, the issue of fiber size length, width and purity is addressed in part four of the paper.

Q Which may be published in the future?

A Yes.

Q Are you familiar with abstracts that Dr. Kent Pinkerton, Dr. P. Pratt, A. R. Brody and J.D. Crapo published in the "American Review of Respiratory Disease," April of 1981?

A These are the three abstracts we talked about before?

Q Right.

A I'm familiar -- well, you have to show them to me again.

Q Okay. I'm only showing you the one published which I just described.

A Okay.

Q Would you agree with me that in that

abstract, those authors say that all three types of chrysotile, including the Calidria caused interstitial lung injuries after three months of exposure by the rats?

A Can I read this?

Q Can you answer the question?

A I can't remember the question.

MR. BROWNSON: Read it back.

MR. WILL: Let him read the abstract first, and then we can have the question read back.

A Yes, I see his statement here. After three months' exposure, all fibers caused injury to the epithelium and interstitium with the increase of the volume of alveolar type II cells. I don't consider those injuries.

Q So even though they call them injuries, you don't consider them injuries?

A No, I don't consider them injuries. I don't see any data, either.

Q Well, you don't see any data; but this are the same rat inhalation studies that you are talking about in your series of three papers, isn't it?

A Right.

Q And you have no reason to believe that

Dr. Pinkerton would make that up, do you?
A I'm just saying that he is citing an abstract and there is no, to my knowledge, published data prior to that point to support his contention; and I don't see any intended data in which he is basing his statement.
Q So if it is your contention, then, that he if makes that statement, he is simply wrong?
A I wouldn't call an increase in epithelium and an increase in interstitium volume necessarily an injury, because we see all that as rats age. That is supported in his thesis, Pinkerton et al. 1982; and Pinkerton's thesis demonstrates the same phenomena.
Q So you believe he was wrong?
A Well, I wouldn't use the word injury.
Q If he again uses the word "injury" in another of his abstracts, would he be wrong again?
A It depends. Show me the abstract.
Q I show you the abstract published in the "American Review of Respiratory Disease" at the annual meeting of the American Lung Association, the Canadian Lung Association, the American Thoracic Society, the Canadian Thoracic Society and the Congress of Lung Association Staff in 1980.
I have highlighted the sentences there.

A Could we make a copy of this?

Q Before we do, can you answer the question? In that abstract, again, Dr. Pinkerton describes what the lungs received from exposure to all three types of asbestos as injury, doesn't he?

A Yes, he refers to them again as injury and my response would be the same again. He makes the same statement, volume and thickness of epithelium and interstitium increase, alveolar cell increase.

I don't think that's necessarily indicative of injury.

(A short recess is taken.)

Q Going back to your paper, Dr. Ilgren, part three that we have been talking about today, page 102 in the second column at the bottom of the first paragraph you write, "The average density of Coalinga fibre in interstitial cells at three months appeared to be three times greater than UICC/B and Jeffrey, but this was due to one animal with an anomalously high value, so the difference has probably been significantly overestimated."

Do you see that?

A Yes.

Q So what you are saying is the references or the data we see on table 3 of your paper showing

that the average density of the Coalinga fiber in the interstitial cells at three months to be greater than the other two was due to one anomalous animal?

A Yes. It's 21.8, isn't it?

Q If we look at table 3, can you show us where that reference appears?

A The reference or the animal?

Q The anomalous animal.

A I think it's 21.8. I just mentioned it.

Q Where do we see that?

A Look here. If you look at table 3, you go to the column Coalinga and go under alveolar macrophages; and then you go to three months and you see 8.52 plus or minus 4.69 then in brackets you have 0.5-21.8.

So I'm referring to that animal, 21.8. If you look at the individual -- I don't have the raw data here, but the 21.8 seems to be a major outlier, greater than the other points.

Q Now, you had told us at an earlier deposition that all of these animals in the raw data from Dr. Pinkerton that you looked at had a number to them.

A Right.

Q Can you tell us the number of that

anomalous rat?

A I could. I can't sitting here, but I could do that.

Q That data is available to you?

A Yes.

INSERT:_____

Q You described that as an anomalously high value; but however you described it, that is an actual value in a rat, right?

A Right.

Q In other words, what that indicates is that there is one rat that breathed a lot of Coalinga fiber and for some reason a lot of it -- or there was a lot of fiber density change in that particular rat.

MR. WILL: Object to the form of the question. Why don't we just stick with the fiber density change?

Q One rat had a large fiber density change following exposure to Coalinga?

A It had a fiber density, yes.

Q And although you describe that as an anomalous rat, that is a piece of real data?

In other words, that rat had a lot of Coalinga fiber in its lungs, didn't it?

A Right.

Q I take it you don't know why that particular rat had so much Coalinga in its lungs?
A No.
Q Could it be particularly susceptible or the fact that it was so stupid it kept its mouth opened when it breathed? It could be any number of things, right?
A I imagine so. I don't know why.
Q Now, if you look at page 103 of your paper in the second column about the bottom third you again speak about an extremely high fiber density in one Coalinga animal.

Is that the same one? That's the same one that we were talking about on page 102 or a different one?
A Let me check here. That's the same one, I think.
Q Because now we are talking about fiber density in the alveolar macrophages.
A I beg your pardon, I made a mistake. When we were talking about the anomalous value on page 102, that had to do with cellular interstitium and that would be cellular interstitium; and that would be, again, if you look at the table 2 on page 101 under interstitial cells.

Q Now, is this table 3 or table 2?
A Wait a minute, hang on. I'm sorry.
Q Take your time, because I'm trying to figure out who these anomalous rats are?
A With reference to page 102 and our discussion just a minute ago where you were reading the average density of Coalinga fiber in interstitial cells in three months appeared to be three times greater than UICC/B and Jeffrey, but this was due to one animal with an anomalously high value. So the difference has probably been significantly overestimated, I should have been referring to the portion of page 101 in table 3 under interstitial cells for a three-month value of 1.98.
Q So is the anomalous rat described at page 102 of your paper seen in table 3 under interstitial cells at 1.98?
A Yes.
Q And what is the anomalous rat described at page 103 of your paper?
A That is the one that I formerly described as under alveolar macrophages at three months referring to 21.8.
Q So if we can summarize, the average density of Coalinga fibers in the interstitial cells

at three months is much higher in the Coalinga; but you attribute that to an anomalous rat, right?

A Yes.

Q At least in part?

A Yes.

Q The fiber density leading to an increase in the number of alveolar macrophages is, again, higher than the Coalinga; but you attribute that to another anomalous rat?

A Maybe.

Q It might be one reason for it?

A Might be.

Q Whatever the reason for it is, however, the fact remains that the number of alveolar macrophages is increased more in the Coalinga than the UICC/B at three months?

A Right.. It's not the number it's --

MR. GERSON: Off the record.

(A discussion is held off the record.)

A Are you referring to number of macrophages or are you referring to fiber density in macrophages?

Q I don't know. You are calling it number of macrophages in your paper?

A No, in table 3 it is not the number. The number of macrophages is table 7. Table 3 refers to

the density of fiber in macrophages, the number of macrophages.

Q That's not what I'm talking about.

MR. WILL: The answer to your question is no. That's what we are getting at.

MR. BROWNSON: Let's go back and start over.

Q I'm reading at page 103 of your paper. Under the heading of alveolar macrophages, that says there was an increased number of alveolar macrophages in the Coalinga versus the UICC/B; does it not say that?

A Show me exactly where you are reading from.

Q Let me quote --

A No, just tell me in regards to where you are.

Q Right here.

A Inhalation of Jeffrey and Coalinga fiber for three months led to a greater than 84 percent increase in the number of alveolar macrophages, compared with age-matched control animals, citing table 4.

MR. WILL: But you were talking about table 3.

MR. BROWNSON: We were talking about something else then. Please, we need to

listen closely to the questions.

MR. WILL: Let's go off the record for a second.

(A discussion is held off the record.)

MR. WILL: Dr. Ilgren, let me ask you to clarify. Does table 3 refer to the number of macrophages?

THE WITNESS: No.

MR. BROWNSON: I never said it did. Please listen to the question.

Q What does table 3 refer to?

A Fiber density.

MR. WILL: In the macrophages?

THE WITNESS: Yes.

MR. BROWNSON: Now we have to start over.

Q The first anomalous rat described in your paper is at page 102, correct?

A Right.

Q And that anomalous rat, in your view, contributes or causes the increase in the average density of Coalinga fiber in interstitial cells at three months?

A Yes.

Q As shown in table 2?

A No, table 3.

Q Table 3?

MR. WILL: Because table 2 is volume changes.

Q Table 3 of your paper at page 101, right?

A Right, and the anomalous animal there is 1.98 at three months and table 3 on page 101 under interstitial cells.

Q Now, the second anomalous animal you describe in your paper, you describe at page 103, right?

A Right.

Q And that second anomalous rat caused the difference in the number of alveolar macrophages at three months of exposure?

A No. The anomalous value referred to on page 103, as far as I can see, refers to fiber density, not cell number.

If you look on column two, page 103, five lines up from the bottom it reads, "However, much of the difference between UICC/B and Coalinga was due to an extremely high fiber density reading of one Coalinga animal."

Q Now, I will ask again, is that the same anomalous rat as we were just talking about described on page 102 or is that a different one?

A That, I don't know.

Q You will agree with me, at least, that the anomalous rat described on page 103 is extremely confusing, because it's under the heading of alveolar macrophages when you are talking about the increase in number of them; but then when you get to this anomalous rat, now we are suddenly talking about fiber density.

MR. WILL: I object to the question. It's argumentative. It's speculative and it's multiple.

A I don't understand the question.

Q Under your heading of alveolar macrophages on page 103 the first sentence reads, and I quote, "Inhalation of Jeffrey and Coalinga fiber for three months led to a greater than 84 percent increase in the number of alveolar macrophages, compared with age-matched control animals (table 4). However, exposure to UICC/B did not increase macrophage numbers significantly by this time."

Then you continue and down later in that column we get to this anomalous rat.

A But it's apples and oranges again. The beginning of the discussion on page 103 about alveolar macrophages talks about cellular number and

cellular volume changes. Later on page 103 we are talking about fiber density.

Q Is that the same anomalous rat as you were talking about on page 102?

MR. WILL: He told you twice now. He doesn't know. Asked and answered.

A The only way I can tell is if you put out the raw data for all four or five animals.

Q And your paper doesn't tell you which anomalous rat this is?

A No, just the raw data.

Q Is it fair to say, Dr. Ilgren, that all three fiber types, including the Coalinga, caused increased macrophage cell volume after three months, as described in table 4?

A That's what I have said in my paper.

Q However, the density of the asbestos in macrophages was higher for the Coalinga than the UICC/B, correct?

A I just want to be sure I get your question. Ask it again.

Q The density of Coalinga fiber in the rat macrophages was more than twice the amount than after the exposure to the UICC/B fiber, correct?

MR. WILL: The Coalinga was more than

twice the UICC/B?

MR. BROWNSON: That's what I said.

MR. WILL: You didn't say which one.

MR. BROWNSON: Stop. Let me ask a question. Listen carefully to the question.

MR. WILL: I was. That's why I interrupted.

Q Listen carefully to the question. The density of the Coalinga fiber in the macrophages was more than twice that of the UICC/B fiber in the macrophages?

A Yes.

MR. WILL: At three months?

MR. BROWNSON: At three months.

Q And you attribute that density difference or much of it to one anomalous Coalinga rat?

A Yes.

Q Again, we don't know which Coalinga rat that was?

A Right.

Q But that is a real piece of data?

A Sure, it's a real piece of data.

Q Now, then turn to page 104 of your paper. In the first column at the last sentence of the first full paragraph, you again are describing anomalously

high fiber density in a Coalinga rat.

Do you see that?

A Yes.

Q Now, which rat is that one?

A Again, it's the same, unless I have the raw data, I can't really identify which one it is.

Q Well, let me point out that here you are talking about the density of Coalinga fiber after twelve months' exposure.

A Right.

Q So can you tell from that if this is that same anomalous rat we had seen earlier or if this is a different one?

A No, we can't tell.

Q At twelve months' exposure, this particular anomalous rat described on page 104 had density of Coalinga fiber in its lungs more than six times greater than that found in the Jeffrey treated macrophages, right?

A Right.

Q Let me ask you this question, Dr. Ilgren. Your paper at page 104 says that the density of Coalinga fiber after twelve months' exposure appeared to be more than six times greater than that found in the Jeffrey-treated macrophages, right?

A Are you talking about the average of the Jeffrey?

Q I'm quoting directly from your paper. I quote, "Although the density of Coalinga fiber after twelve months' exposure appeared to be more than six times greater than that found in the Jeffrey-treated macrophages, this was largely due to one exceedingly high, anomalous fiber density reading in a Coalinga-treated animal (table 3)."

Do you see that?

A I see it.

Q Can you show us which animal that is in table 3?

A You want me to tell you which is the anomalous reading that I'm referring to?

Q Yes.

A Well, I would say it's the 23.8 reading.

Q And that is under the heading Coalinga in the first section at twelve months under alveolar macrophages?

A Right.

Q So if I could summarize, Dr. Ilgren, because I circled them as you gave your answers, there appear to be three anomalous rats here. The first is --

A You can't say that.

Q Well, let me just finish. The first is that under alveolar macrophages three months' exposure in your table 3 at 21.8; the second one is at twelve months' exposure at 23.8 and the third is three months' exposure under interstitial cells at 1.98.

MR. WILL: Objection. He said there is three anomalous readings, not three anomalous rats.

He told you he couldn't answer the question. Don't answer it. Let him fix it.

Q There are three anomalous readings. They may or may not be one rat, two rats or three rats; is that correct?

A That's correct, but that's not what you asked me.

Q And you don't know if it was one rat, two rats or three rats?

A That's correct.

Q Nor do you set forth any data information or rat number in any of your three papers to tell the reader this is one anomalous rat, two anomalous rats or three anomalous rats?

A That's correct.

Q Nevertheless, it is upon this one, two or

three anomalous rats that you report that the Coalinga results are higher both at the three-month alveolar macrophages, the twelve-month alveolar macrophages and the three-month interstitial cells?

A Right.

Q Now, I would like to turn to page 105 of your paper. On the first column about two-thirds of the way down, you are talking about this 223 million fibers that Dr. Davis found in that study that we talked about earlier?

A Dr. Muhle.

Q Dr. Muhle?

A Yes.

Q And you say that a substantial number of fibers, 223 million, noted twelve months post exposure in Dr. Muhle's inhalation study seems to be at odds with some observation.

What observation was that?

A Six lines above that I note the very low (0.03 plus or minus 0.03 mg) post-ashing fiber mass readings observed by these workers, which is Muhle et al.

Q So in Muhle's rat inhalation study, when he did this fiber ashing to come up with the asbestos mass in the lungs, he got a low value; but when he

actually counted fibers, he got his 223 million; and you are saying that there seems to be some odds between those two things?

A Right.

Q And then you continue and I quote, "The most likely explanation for these apparently discordant findings is probably a trivial technical one," right?

A Right.

Q What do you mean by trivial technical explanation?

A Well, if you read on --

Q I did read on but I couldn't figure it out.

A Well, what I'm saying is that the 223 million all sizes or eleven times ten to the sixth is greater than five microns. The number is probably -- when you look at that with TEM, it's actually a relatively small number.

Stated differently, it may also be that since long fibers contribute more to mass than short fibers, then what he is actually measuring as long fibers may not be long fibers.

Q But when he is measuring them by TEM and counting fibers, he is actually seeing them. That

was not a mass calculation, was it?
A I know but it goes to the question as to what he is actually counting, even by TEM, as a long fiber.
Q Well, presumably, he is counting fibers, because that's what people do with TEM, isn't it?
A Well, one counts structures and the structure may be a complex structure or it may be a single. I don't think you can tell by this.
Q Whatever it was that Dr. Muhle saw under this TEM, he reported it in his paper as fibers, right; and you are now speculating, well, it might have been something else?
A Well, let me read through this for one second and then I will return to your question.
I think what's going on is there is a discrepancy between what appears to be a very low mass and a high fiber number; and I think one explanation for that is that, as Muhle points out and he says here, "considerable decrease of fiber diameter observed for the Coalinga fibers in lungs compared with fibers in the inhalation chamber, which occurred because of splitting. The splitting produced fibrils that were, in fact, too fine to count and size reliably."

Q Right, which he documented under his TEM, correct?

A Right, but the fibrils have very, very little mass, per se. So I think what's happening is that the very low mass dose does not necessarily reflect the increase or this number of fibrils.

Q In the Muhle study the Coalinga fibers seen under the TEM, Muhle thinks, might have been much thinner, which attributes to their smaller mass; is that what you are saying?

A Yes.

Q But nevertheless, the 223 million fibers that he points out in his paper are counted fibers, although they are thin?

A Right.

Q Going back to the anomalous rats whether it's one, two or three, do you know if any of those rats, rat or rats, were the ones that got tumors, as you read in part 2 of your paper?

A I would have to go and check. You mean, of the one or two tumors that came in the Coalinga animals?

Q Part 2 of your paper is about the tumors and some of the Coalinga rats had tumors.

My question is, do you know if any of the

tumors in the Coalinga exposed rats were in those rat or rats described as the anomalous rats in part 3 that got the high Coalinga fiber?

A No. You mean just sitting here?

Q Can you tell by looking at the papers, because paper 2 does actually list rats by rat number?

A I can't because I don't have the correlated data in this paper.

Q In the abstract of paper number 3, I'm going now to the abstract on the first page, on the fourth line -- actually, the third and fourth line you write, "A short amphibole-free chrysotile from Coalinga, California failed to induce pathological effects whilst the other two, both long fibers from Canada, were both fibrogenic and tumourigenic."

Do you see that?

A Yes.

Q And you say you have demonstrated that in papers 1 and 2?

A Yes.

Q Again, if you look at paper 2 table 1, the Coalinga induced rats had two tumors and three BAH, right; and we talked about that before?

A Yes, we have gone through that.

Q Basically, in order for the statement in the abstract part 3 to be correct, there shouldn't be the two tumors and the three BAHs in table 1 of part 2.

A No, there were two tumors in the controls. I have subsequently added that to be two carcinomas looking at the raw data. I don't think there is a significant increase in tumors above the controls.

I don't think there is any difference between the number of tumors in the controls or the Coalinga-treated animals.

Q We have gone through that at length. We will let that stand. We won't get into it again.

MR. BROWNSON: That's all I have.

MR. WILL: I have a couple questions.

CROSS-EXAMINATION BY MR. WILL:

MR. WILL: Mark these please.

(A copy of "American Review of Respiratory Disease" Volume 123 dated April 1981 is marked as Plaintiff's Exhibit 45 for identification, as of this date.)

(A copy of "American Review of Respiratory Disease" Volume 121 dated April 1980 is marked as Plaintiff's Exhibit 46 for

identification, as of this date.)

Q Dr. Ilgren, the first thing I want to ask you is, you were asked a question by Mr. Brownson about whether the anomalous rat at twelve months in terms of the fiber density and the alveolar macrophages that had been exposed to the Coalinga fiber whether that rat more than six times increased density compared to the Jeffrey; do you remember that question?

A Yes.

Q When you said six, is that comparing the averages of the two exposed groups?

MR. BROWNSON: Objection, leading. You already clarified that.

MR. WILL: That was a different question.

MR. BROWNSON: I will agree with you. It was not the anomalous rat that had six times more. It's the Coalinga group which had six times more, which he attributes largely to that anomalous rat.

MR. WILL: That's not the question you asked.

Q Let me put it this way. Look at table 3, alveolar macrophages. If you compare the average for the group Coalinga versus the average for the Jeffrey

group, is that where you get the six times difference?
A At twelve months?
Q Yes.
A Yes.
Q Obviously if we were comparing the relationship between the value for the anomalous rat, we could get a different number than six?
A Yes.
Q You were asked whether the three so-called anomalous readings came from the same rat or different rats.

What, if anything, can you tell about that question, based on the dates of the readings?
A You mean three, twelve or twenty-four months?
Q Three or twelve months, yes?
A Well, the correct answer is that it would have to be at least two rats, because you wouldn't have the same rat at three months and twelve months. They are killed at three months.
Q And with respect to the two rats that were found to have the tumors, do you know anything about what their sacrifice date was?

MR. WILL: I'm going back to page number 2, Mr. Brownson.

Q Do you know anything about the sacrifice

date of those rats, just based on the fact that they had tumors?

A I don't believe any of the rats had tumors at three or twelve months. So it couldn't have been either of those.

Q So based upon those assumptions, the anomalous rats would not be the ones that developed tumors?

A That's right.

Q Let me show you what I have marked as Exhibit 45. Can you tell me briefly what that is?

A This is the abstract by Pinkerton et al., lung injury patterns in rats.

Q And what's the date of that one?

A April 1981.

Q Is that the document Mr. Brownson showed you earlier?

A Yes.

Q Let me show you what I had marked as Exhibit 46 and ask you what is that?

A This is morphologic lung changes following exposure to chrysotile asbestos fibers by Pinkerton et al. published April 1980.

Q Now, Mr. Brownson asked you some questions about the statements in the abstracts by

Dr. Pinkerton that refer to injury.

Do you recall those questions?

A Yes.

Q Why is it that you would not call the reported changes an injury?

A They are not irreversible. There is no evidence that they are irreversible, nor is there clear evidence that they manifest overt fibrosis and tumor formation.

Q Are you doubting that Dr. Pinkerton, in fact, observed the characteristics that he reports in the abstracts?

A Not at all.

Q Have you observed and reviewed the same tissue slides that he is talking about here?

A Yes.

Q Did you observe the changes or the conditions that he remarks upon?

A Well, let's step back. When you say I observed the same slides and the same conditions, I mean, I understand he is saying there is injury based on an increase in value and number of alveolar type II cells and an increase in interstitial matrix after three months; but I didn't look at any of the materials under a TEM microscope. So I haven't, to

that extent, looked at the material, no.

Q Did you look at them under an SEM microscope?

A Yes -- no, actually after three months there was no available material.

Q Was there after twelve months?

A No, there was available for end of life.

Q To get back to where I was, are you disputing that Dr. Pinkerton saw the physical characteristics that he reports in the abstract?

A No.

Q How would you characterize those physical characteristics?

MR. BROWNSON: Well, that's been asked and answered.

THE WITNESS: I don't think that's been asked and answered at all.

MR. BROWNSON: Well, go ahead and answer it; but I think it has been.

A I think the reactions to the presence of an irritating material administered to animals in very high doses for a long time.

Q Is that reaction or response specific to asbestos?

A Not at all.

Q Let me turn your attention to Exhibit 46 to the early part of the abstract; and there is a statement in it that says, "Greater than ninety percent of the fibers in both groups are less than five microns in length, but the mean fiber mass in the intermediate range fibers is 45-fold greater due to the presence of a larger number of long fibers in that chrysotile preparation."

Did I read that correctly?

A Yes.

Q What do you understand the intermediate range fibers to be?

A Jeffrey.

Q What about the UICC/B, what do you understand that to be?

A That's not here.

Q How does the concept of fiber mass relate to the concept of fiber number, if at all?

A Well, long fibers contribute far more to mass than tiny small fibers.

Q Does the fact that an animal got a greater mass of Jeffrey fibers than Coalinga fibers necessarily mean that the animal got more Jeffrey fibers than Coalinga fibers?

A Well, as stated, it suggests that the animals

got a larger number of long fibers.

Q As you understand it, did the animals receive a higher mass of Jeffrey asbestos than Coalinga asbestos?

A No, it's stated right here. They got three times higher mass dose of Jeffrey than Coalinga.

Q The fact that they got three times higher mass dose of Jeffrey than Coalinga, does that mean that they got three times as many fibers of Jeffrey than Coalinga?

A You mean long fibers or short fibers?

Q All sizes.

A Presumably, yes. Presumably that if they got a greater mass dose, they would have gotten a greater number of fibers of all sizes.

Q Does that depend upon the mass of each particular fiber?

A Yes.

Q So it's possible to have more fibers of Coalinga if they are smaller in mass, as we were talking about before?

MR. BROWNSON: Objection to that last question as leading.

You can't impeach your own witness.

A I think what one has here is --

Q Doctor, I just want you to answer my question. The question is, does the size of the mass necessarily dictate the number of fibers?

A No.

Q Is it possible to have more fibers in a smaller mass of asbestos than in a larger mass of a different kind of asbestos?

A Yes.

Q Can you tell from the data here whether there were necessarily more Jeffrey fibers or more Coalinga fibers?

MR. BROWNSON: That's been asked and answered.

A No.

Q Directing your attention to the last five lines of Exhibit 46. The sentence there beginning on the fifth line says, "In addition, light microscopy revealed a greater degree of fibrosis and hypercellularity in the perivascular and peribronchial interstitium of the animals exposed to intermediate range fibers."

Did I read that correctly?

A Yes.

Q What type of fiber do you understand that to refer to?

A Jeffrey.

Q In terms of the reaction, response in this rat tissue that you described earlier, is it possible that some of that is reversible?

A Sure. Well, that's what the data showed, resolution. We have evidence of reversibility.

Q With which type of exposure?

A With Coalinga, largely with Coalinga and there is a certain degree of resolution with all three types; but it's most marked with Coalinga.

Q You were asked some questions about your characterization of the Coalinga as a short fiber.

Do you recall that?

A Yes.

Q What do you mean in terms of characterizing asbestos as short?

A The size cut off.

Q I'm asking what you mean when you talk about asbestos as being short. What do you mean? What's your definition?

A Less than five to eight microns in length.

Q Does that mean that every single fiber in the sample would be less than five to eight microns?

A No.

Q What does that mean then?

A A predominance, something is predominantly short.

Q The fact that there may be some Coalinga fiber longer than five to eight microns, does that suggest to you that your characterization of it as short is an error?

MR. BROWNSON: I object as leading.

A No. In direct preparation it's clear. There is some there.

Q You were asked some questions earlier about this Muhle study that used TEM for the twenty-four month counting while using SEM for the prior count; do you recall that?

A Yes.

Q What is the difference in sensitivity or resolvability, if you will, of TEM as opposed to SEM?

A It's my understanding that with SEM you can't resolve things that are less than 0.2 microns in width, whereas with TEM, these can be seen.

Q In terms of a comparison of the numbers of fibers that you could observe with TEM versus SEM, is there a standard factor or conversion number?

A My understanding in a very, very general sense would be something like ten to a hundred to one; but again, I would have to check that.

Q It would be ten to one hundred fibers visible under TEM compared to one visible under SEM?

MR. BROWNSON: Object to the form of the question as leading. He also said he didn't know.

Q Is that your understanding?

A Yes. If Muhle is saying that the splitting is causing most of the Coalinga in the lung to become fibrils, fibrils are going to be less than 0.2 microns in width.

Q You were asked some questions earlier this morning about the Waggoner case out in California and you were asked about whether policeman Waggoner was alleged to have any asbestos exposure, other than what he got from driving around in his police car. Do you recall that?

A Yes.

Q To your understanding, what were the other asbestos exposures that policeman Waggoner was alleged to have had?

A Manipulating or cutting transite pipe, working with drywall and other construction-related exposure, either directly or indirectly. I don't recall exactly.

Q Do you have an understanding of whose

transite pipe that was?
A I think it was JM, but I can't remember.
Q Assuming that it was JM, do you have knowledge about what type of fiber was used in JM transite pipe?
MR. BROWNSON: Do you know this, Dr. Ilgren?
THE WITNESS: Yes, I do actually.
MR. BROWNSON: He doesn't know what exposure this policeman had.
THE WITNESS: He didn't ask me what the exposure the policeman had. He asked me about a product composition that is in various Answers to Interrogatories in certain cases.
A Chrysotile is not an uncommon component of JM transite pipe.
Q From your own personal knowledge, do you know where that chrysotile came from?
A Not infrequently from Australia.
Q From the Wittenoom deposit?
MR. BROWNSON: Objection, leading.
Q Where in Australia, then?
A The Wittenoom deposit.
Q You were asked a question by Mr. Brownson regarding the mineralogical composition of

chrysotile. I know you are not a mineralogist.

MR. BROWNSON: You are disqualifying him right from the question.

Q I'm not trying to ask you a terribly technical question.

What is your understanding about whether all chrysotile are the same mineralogically?

MR. BROWNSON: Objection to the form.

A They are not.

Q Is there anyplace in your paper where you, in either of the three papers, where you indicate what length cut off you were using for long versus short?

A Yes.

Q Where is that?

A I think it's in the beginning of the part 1 or part 2. I think it's actually in the abstract or perhaps in the fibrosis paper.

Q Is that part 1?

A Part 1.

Q I'm showing you a copy of part 1, which was previously marked as Exhibit 36.

A Well, I refer to greater than five to eight microns in length.

Q As being long?

A Yes.

MR. GOLDMAN: What line and what page?

THE WITNESS: Line 3, page 265 part 1.

Q In the scientific literature, are there papers that characterize Coalinga as being either long or short?

A Yes.

Q What is the characterization in the literature?

A That it's a short preparation.

Q Can you identify some authors that have characterized Coalinga as a short fiber?

A Langor et al. 1978; Yaeger, et al. 1983; Campbell et al. 1980; Siegrist and Wyle 1979; Wyle 1980, among others.

MR. BROWNSON: Name the others.

THE WITNESS: I can't recall at the moment.

Q Those are the ones that you can recall off the top of your head?

A Yes.

Q First of all, we marked as Exhibit 45 and 46 two of the Pinkerton abstracts, correct?

A Yes.

Q Are there any other Pinkerton abstracts?

A There is one other. I think there are three.
Q Do those abstracts present the set of data that you do in your three papers?
A No.
Q Do the abstracts present any data at all between the twelve and twenty-four month period?
A I have to look.
Q Fine. We won't go back and review that. You don't recall?
A I don't recall. I think one might, but I don't recall.
Q We can look at them to see, right?
A Yes.
Q Mr. Brownson asked you with respect to your earlier papers 1 and 2 about the differing numbers of the rats that were on tests; do you recall that?
A Yes.
MR. BROWNSON: Was that today I asked that?
MR. WILL: No, you asked him that the last time; but since I never had a chance to ask questions the last time, I'm asking now.
Q And the question I have for you is, how many sources of data are there for giving numbers for

rats on tests?
A Half a dozen, at least.
Q Are those different sources of data consistent in terms of the number of rats they report?
MR. BROWNSON: Who reports? I'm confused.
A I mean, there is inconsistencies in the reporting.
Q Do you have this data somewhere in your files?
A Yes.
Q Did you have it with you at the deposition when Mr. Brownson was asking you about whether it was 320 or 330 or 360 rats?
A No.
Q When you were answering the questions from Mr. Brownson last time, were you going, then, as best as you could from your memory?
A Yes.
Q I want to direct your attention to table 1 of your paper number 2, which was previously marked as Exhibit 37. This reports animals with tumors; is that correct?
A Yes.

Q Was this data previously reported in 1984 by the NIEHS publication?
A It was presented by the NIEHS for the untreated controls and for the UICC-treated animals.
Q Not for the Coalinga or the Jeffrey?
A Correct.
Q As for the controls and the UICC animals, are the same slides and the same animals presented in your table 1 that were presented by the NIEHS in 1984?
A Yes.
Q Let me direct your attention to table 8 of paper 1, which was Exhibit 36. In terms of the rats that are represented in table 8 of paper 1, is it your understanding that there were eighty rats there?
MR. BROWNSON: Where?
A Eighty rats where?
Q Did you look at the --
A I looked at Coalinga COF-25.
Q There are ninety rats in that group, correct?
A As stated there, yes.
Q I know you said earlier you did not look at the slides of the animals that Pinkerton killed or

sacrificed at three, twelve and twenty-four months, correct?

A They weren't available.

Q And those are the rats that were looked at for the morphometric analysis?

A Yes, and for morphological analysis, for both.

Q To your knowledge, was any tumor reported in any of those animals that were sacrificed at three, twelve or twenty-four months and were exposed to Coalinga?

A No.

MR. WILL: Give me two minutes and then I think I'm done.

(A short recess is taken.)

Q Dr. Ilgren, I want to ask you, putting aside for a moment the question of comparing TEM to SEM counts and just taking the numbers at face value as they are reported in Muhle's paper, in your opinion, what, if anything, does that data show about the clearance of Coalinga chrysotile?

A The mass data shows it's completely gone. There is a small residue.

MR. BROWNSON: 223 million fibers.

THE WITNESS: I don't consider that reflective at the moment.

MR. WILL: He talked about mass not fibers.

Q But aside from Mr. Brownson's interruption of your answer, you were talking about mass. Are you finished with that?

A Yes.

Q And even if we look at number of fibers, again, forgetting about the difference between TEM and SEM, what does Muhle's data show with respect to the number of fibers?

A Well, you have apples and oranges. You look at them using one technique and you will see a radical drop-off between the twelve and twenty-four time point.

Q Even using different techniques, do you see any change over that period?

A You do.

Q I want to ask you this, Dr. Ilgren, to a reasonable degree of medical certainty, in your opinion, what does all of this data set forth in paper 1, 2 and 3 mean, if anything, about Coalinga fiber?

A It doesn't -- there is near total clearance. So it doesn't biopersist and that accounts for the fact that it doesn't induce fibrosis in tumors.

MR. GERSON: It being the Coalinga fiber?

THE WITNESS: The Coalinga fiber.

MR. WILL: Subject to anything Mr. Brownson may have for you on recross, that's all I have. Thank you.

MR. BROWNSON: I have nothing further.

(Time noted is 4:35 p.m.)

C E R T I F I C A T E

I, EVAMARIE WALSH, a Notary Public and Shorthand Reporter, do hereby certify that prior to the commencement of the examination

DR. EDWARD ILGREN

was sworn by me to testify to the truth, the whole truth and nothing but the truth.

I DO FURTHER CERTIFY that the foregoing is a true and accurate transcript of the testimony as taken stenographically by and before me at the time, place and on the date hereinbefore set forth.

I DO FURTHER CERTIFY that I am neither a relative of nor employee nor attorney nor counsel for any of the parties to this action, and that I am neither a relative nor employee of such attorney or counsel, and that I am not financially interested in the action.

Notary Public

C E R T I F I C A T I O N O F W I T N E S S

I have read the foregoing transcript of my deposition and find it to be true and accurate to the best of my knowledge and belief.

_____

DR. EDWARD ILGREN

Sworn and subscribed to before me on the _____ day of _____, 1999

Notary_____

My Commission Expires _____