IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF PENNSYLVANIA

In Re: Asbestos Products Liability Litigation (No. VI)	No. MDL 875

UNITED STATES DISTRICT COURT
FIFTH DIVISION
DISTRICT OF MINNESOTA

CONWED CORPORATION, Plaintiff, -against- UNION CARBIDE CHEMICAL AND PLASTICS COMPANY, INC., (f/k/a UNION CARBIDE CORPORATION), Defendant.	Case No. 5-92-88
UNION CARBIDE CHEMICALS AND PLASTICS COMPANY, INC. (f/k/a UNION CARBIDE CORPORATION), Third-Party Plaintiffs, -against- OWENS-CORNING FIBERGLAS CORPORATION, WALKER JAMAR COMPANY, A.W. KUETTEL & SONS, INC., API, INC. and MacARTHUR COMPANY, Third-Party Defendants.	

November 18, 1994
EDWARD B. ILGREN

November 18, 1994
9:45 a.m.

Continued deposition of EDWARD B. ILGREN, taken by Plaintiff, pursuant to adjournment, held at the offices of Kelley Drye & Warren, Esqs., 101 Park Avenue, New York, New York, before David Ocanas, a Certified Shorthand Reporter and Notary Public within and for the State of New York.

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Also Present:
VIRGINIA RUSZCZYK

oOo

EDWARD B. ILGREN, resumed, having been previously duly sworn, was examined and testified further as follows:

MR. BROWNSON: We're back continuing your deposition in the case of Conwed versus Union Carbide. And this is a continuation of the prior deposition in this case.

I want to start with a few preliminary matters that hopefully we can dispense quickly. The issue of the documents we requested at the last deposition. I understand you made a search for and have been able to produce some things and maybe not some other things. What I would like to do is ask you what you looked for, what you produced and what you didn't produce. And maybe the easiest way is for me to go through my list of things, unless you think it might be easier for you to explain to us what you did. I'll take suggestions from you.

MR. WILL: Do you have the list we provided you? That would probably --

MR. BROWNSON: The list you provided? No.

MR. WILL: You got the Federal Express documents.

MR. BROWNSON: I got the box, but there wasn't a list.

MR. GERSON: There was a list in mine.

MR. BROWNSON: Off the record.

(Discussion off the record)

(Whereupon, document entitled "Unpublished Materials & Correspondence Pertaining to Calidria Reviewed for Conwed Versus UCC by Dr. E.B. Ilgren as of 10 November '94" marked Plaintiff's Exhibit 13 for identification, as of this date.)

(Recess taken)

EXAMINATION BY

MR. BROWNSON:

Q. Now we've marked as Deposition Exhibit 13, an alphabetical typewritten list, looks like you prepared, of various publications and documents. Is that what this is?

A. Yes.

Q. And it's entitled "Unpublished Materials & Correspondence Pertaining to Calidria Reviewed for Conwed Versus UCC by Dr. E.B. Ilgren as of 10 November '94."

A. That's correct.

Q. And I guess can we take that to mean what it says, these are things you have reviewed in connection with this case as of a week ago?

A. As stated, yes.

Q. Have you reviewed anything else since November 10 that pertains to this case or any opinions you intend to give in this case?

A. Off the top of my head, no. But there may be other things that just don't spring to my mind.

Q. If there were other things, would those be in the nature of published things or manuscripts as opposed to specific Conwed documents?

A. I believe so, though I'm not 100 percent sure.

MR. WILL: I don't know whether you're categorizing generally, but I think Dr. Ilgren has seen since then the portions

of Fred Bergstrom's deposition, and James Ministo's deposition and the U.S. G sample survey that was marked in Ken Brule's deposition.

MR. BROWNSON: Just to complete that point, you've seen portions of James Ministo's deposition in his Ministo versus Union Carbide case?

A. I presume so.

Q. Portions of a deposition of Fred Bergstrom, do you know which deposition that was?

A. I don't recall.

Q. Do you know what was the topic or subject matter of the portions of that deposition that you reviewed?

Q. Could you ask that again?

Q. What was the subject or topic matter of the Bergstrom transcript that you reviewed?

A. This I recall where Bergstrom worked in the Conwed facility, what he worked with, whether the conditions under which he worked were dusty or not.

Q. Have you been asked by Union Carbide or lawyers for Union Carbide to review the

MR. BROWNSON: What files?

MR. WILL: His files.

A. You asked us to bring --

Q. I asked you to bring the records you reviewed.

A. That's it.

Q. Did those files contain anything other than records you reviewed? Do they contain letters or reports that you generated?

A. There are a few annotated notes here about different aspects of the case, but there are no individual reports here.

MR. WILL: I think what they tend to be are just excerpts from the records, just put in one place.

Q. For instance I'm looking at your file on Stanley Fleisch. The file contains some medical records, a letter from the Mayo Clinic, a letter to myself, Dr. Mark Wick, University of Minnesota from the Duluth Clinic to Mr. Clark's lawyers. Some treatment record from the Duluth Clinic with history and that sort of thing. And a pathology report from a pathologist from the Duluth clinic. In addition to those records, it

contains some notes which looks like things that you have generated. I'll just show those to you and ask you, first of all, are these things that you generated or were those --

A. I generated it.

Q. I take it you have notes of that type that you generated in each of these six files?

A. I've got them here on Broeffle. This is an empty folder. I don't think I did it every single case. There's none in Maki. Let's see. None on Manisto. I don't see any here on Roseth.

Q. Now for the record just so we can identify, you got files which you produced here today. One on Mr. Fleisch, one on Roseth, one on Ministo, one on Maki and one on Broeffle. And then you say you have one on Mr. Bodway but you don't have it here today?

A. I don't believe so.

Q. Now have you reviewed anything else with respect to the medical condition or diagnosis of these Conwed mesothelioma cases.

A. Like what?

Q. Other than what's here in your file.

A. Like slides or anything like that?

A. No. Were any done?

Q. Not that I'm aware. That's why I'm asking you.

A. No.

Q. Have you made any request of Union Carbide or Union Carbide lawyers that you would like to either do that yourself or see that done on any of those people?

A. I would like to see it done, but I haven't made a specific request at this point in time.

Q. Are you aware of any such work that's contemplated or being undertaken at the present time on behalf of Union Carbide?

MR. GOLDMAN: What do you mean by "such work"?

Q. Do you know whether -- as far you as know, is either contemplating or is actually doing lung tissue fibre burden analysis on any of these Conwed mesothelioma cases?

A. Do I know Union Carbide --

Q. Union Carbide or people acting on their behalf.

A. I don't know.

Q. If it has, it hasn't come to your attention?

A. I don't know what Union Carbide thinks.

Q. The question is not meant to be tricky. Have they told you or do you know if --

A. No.

Q. All right.

A. No one has said anything about it being done.

Q. And how about with respect to Mr. Bergstrom, do you know whether there's any such -- lung tissue fibre burden analysis being contemplated on behalf of Mr. Bergstrom's on behalf of Union Carbide?

A. I might be wrong, but I believe he didn't have a post-mortem. I don't think there's any available tissue, but no one has asked me to do such.

Q. Do you have a file with respect to Mr. Bodway?

A. I believe so, yes.

Q. Do you also have a file with respect to Mr. Bergstrom? I guess all you've seen is a

portion of his deposition. Have you opened a file on that?

A. I don't think so, not yet.

Q. Do you plan to do that?

A. Sure, when I get information.

Q. Now you've told us that in your view, one or two or more of these six putative mesothelioma cases among Conwed workers raised questions in your mind, but you can't recall exactly which ones as to the diagnosis?

A. That's right.

Q. Were there any among of those group of six which in your mind were unquestionably mesothelioma that you did not have questions on?

A. Yes.

Q. Do you remember which one those were?

A. I don't remember which ones they were, but there were certainly cases that I think one would accept as confirmed mesothelioma.

Q. As you sit here today, do you remember how many there were?

A. No.

Q. If we looked at your five files here, could you tell from looking at those which ones

you would accept as confirmed diagnoses of mesothelioma?

A. I haven't reviewed them in detail in some time. I would have to go through them again and make some more notes.

Q. Let me ask you this. I note that your files contain -- I didn't look at all of them, some of them contain reports from Mark Wick, a pathologist from St. Louis?

A. Yes.

Q. Are you aware that Dr. Wick was a pathologist that we retained on behalf of Conwed to review these cases?

A. Yes.

Q. With respect to these six cases, would you be in agreement with Dr. Wick, that is in cases where he says the diagnoses of mesothelioma are confirmed? Are those the cases --

MR. WILL: Unless he takes the time to review these records, I don't think that's an appropriate question.

MR. BROWNSON: Let me rephrase it.

Q. Was it the report of Dr. Wick that

you relied on in making the statement that some of these cases were uncontested -- not Union Carbide contested but confirmed mesothelioma, in your view?

A. It was more than his report. It was certainly a contributing factor. There were a lot of different things. I would like to be able to go in detail if we're going to talk about them, just to refresh my memory.

Q. Can you do that here today during the course of this deposition or is that something that would take longer?

A. It would take longer.

Q. But suffice it to say as we sit here today in this deposition on November 18, there are certain of the six cases that you accept as confirmed mesothelioma diagnoses?

A. It's my recollection that's the case.

Q. And do you recall if there was more than one of the cases where that is true?

A. I believe so.

Q. Do you recall if it's more than two?

A. Perhaps. I don't recall exactly.

Q. All right. Now, in addition to

facilities where they may have had amphibole exposure. At least one, I think it was Ministo, discussed how he worked with amosite under very dusty conditions.

Q. Is it your opinion, Dr. Ilgren, that chrysotile asbestos has nothing to do with the mesothelioma in these six cases?

MR. WILL: In general.

Q. In general at this point?

A. No, I don't believe it did.

Q. No, you don't believe it did -- restate your answer.

A. I don't believe chrysotile contributed to the development of any of these mesotheliomas.

Q. Let me ask you some questions about that. What do you base that statement upon, what facts?

A. My sense is that virtually all of the chrysotile is Union Carbide chrysotile and Union Carbide calidria.

Q. It's your view that chrysotile-exposed workers who develop mesothelioma get that disease from something else

other than chrysotile?

A. That's correct.

Q. When you looked at the six cases of Conwed workers, what you were doing was looking to see if they had any exposure to amphibole-type of asbestos?

A. That's correct.

Q. Do you believe that amosite can cause malignant mesothelioma?

A. I do.

Q. Do you believe that any of the mesotheliomas among these these six Conwed workers were exposed to amosite asbestos?

A. Conceivable.

Q. Do you have any firm opinion on that one way or the other?

A. Probably.

Q. What is that opinion?

A. Probably.

Q. Probably that some were or probably that all were or what?

A. Certainly some were.

Q. Do you know which ones?

A. No.

Q. Now do you believe that some of these mesotheliomas among Conwed workers were caused to exposure to crocidolite asbestos?

A. I do.

Q. What is the factual basis for that opinion?

A. To the best of my recollection, I would have to go back and read the details of the case. Both Mr. Ministo and Mr. Bergstrom worked under very dusty conditions with crocidolite in the R&D facility for, I think it was several years, at least a couple of years, and based on my appreciation of the types of levels and exposures and settings in which people who use crocidolite develop these mesotheliomas, I would say their mesotheliomas were due to the crocidolite they used.

Q. Is it your view there has never been a reported case in this country of a malignant mesothelioma caused by exposure to chrysotile asbestos only, free of amphibole?

A. Could you read it back.

(Question read)

A. People have claimed to report them in

the country. I don't necessary accept they have proven that they're because of chrysotile alone.

Q. Whatever case reports there may be of malignant mesothelioma is due to chrysotile asbestos, it's your view that they were not -- it's your view they were caused by something else?

A. I would like to look at the individual papers, that's my opinion. In that question, were you referring to chrysotile free of amphibole?

Q. By chrysotile, I mean pure chrysotile.

A. All right.

Q. I assume that opinion means that in your view, calidria brand asbestos, which we're talking about in this case, is incapable of causing malignant mesothelioma?

A. That's correct.

Q. Do you know why if that's true, Union Carbide has settled the mesothelioma cases arising on the Conwed workers?

MR. WILL: Don't answer that question. It's absolutely not an appropriate question under Rule 408.

MR. BROWNSON: So you're instructing him not to answer?

MR. WILL: Yes.

Q. I asked you a few questions in this regard last time. Let me just finish the point here.

Have you done work on behalf of Union Carbide in any personal injury cases outside of the Conwed workers which involve exposure or alleged exposure to Calidria asbestos?

A. Yes.

Q. In particular I want to focus on mesothelioma cases. Have any of those involved mesothelioma?

A. Not that I recall.

Q. Do you know if any of them involved any other cancer, lung cancers?

MR. GERSON: Or esophageal?

A. Esophageal.

Q. How many cases were there, was that one case or more than one?

A. How many esophageal cases, I think one.

Q. Other than that esophageal case, have

you worked on behalf of Union Carbide on any other cases involving exposure or alleged exposure to Calidria asbestos?

A. I believe there has been three. Curry, Hodges, Holmes.

Q. Which of those three cases was the esophageal cancer?

A. I don't recall right off the top of my head.

Q. Were the other two asbestosis or some other pulmonary problem?

A. I believe one was asbestosis.

Q. Do you recall what the third was?

A. No.

Q. Do you know whether in any of those three cases, there was a lung tissue fibre burden analysis done?

A. No.

Q. By that, do you mean you don't know or it wasn't done?

A. I don't know. The cases settled very quickly, I did very little work on it.

Q. Do you recall whether you ever saw any lung tissue fibre burden analyses in any of

those cases?

A. I don't remember. I don't recall.

Q. Just to finish up on these three cases, do you remember where those cases were venued, in what court?

A. Let's see, at least one was in Texas and the other two, I don't recall.

Q. And do you remember what the exposure to Calidria asbestos was in that Texas case?

A. I don't recall.

Q. Other than those three cases, have you ever done any work on behalf of Union Carbide or on your own reviewing or looking in any way at groups of workers exposed to Calidria asbestos other than the Conwed workers or other than the people out at King City?

MR. GOLDMAN: Other than the three cases we talked about?

MR. BROWNSON: Those were three individuals cases, I'm now asking about groups of exposed workers.

A. Such as?

Q. Well, I don't know, that's why I'm asking the question.

A. It's a bit vague. Give me an example.

Q. For example, Union Carbide sold Calidria asbestos over the years to many different customers who used it in many different settings, whether in plants or factories or oil fields or different things. And what I'm wondering, you've reviewed or looked at these three individual cases you told us about.

A. Right.

Q. In addition you've done some work with respect to the Conwed workers?

A. Yes.

Q. And in addition to that, I know that you've at least looked at in some respects at information dealing with the workers out at the line at King City?

A. Yes.

Q. What this question is, are there other workers anyplace exposed to Calidria asbestos that you reviewed or looked at? Are there any other workers?

A. No.

Q. Are you aware of any sort of

epidemiological study done by anyone dealing with any other group of Calidria-asbestos-exposed workers other than King City, Conwed?

A. Not that I can remember.

Q. I'm not just talking about published work. I think you work -- there is no epidemiological work with Calidria-exposed workers. My question went to unpublished work that you may have heard of or seen.

A. Nothing comes immediately to mind.

Q. Now let's go back to the six Conwed workers.

A. Could you ask the question again.

Q. I'll have the record read back.

(Record read)

A. I don't think so.

Q. I wanted to go back to the six Conwed mesothelioma cases you reviewed. You've told us that one of the purposes of your reviews -- of your review of those six cases was to look at the diagnosis and another purpose was to try to make -- establish some opinion as to the causation of those mesothelioma. Did you review them for any other purpose? Was there any question that

you answered as a result of your review?

A. Not that I'm aware.

Q. With respect to possible crocidolite exposure on those six workers, I understand you identified the use of some crocidolite in the research and development setting with respect to Mr. Manisto and Fred Bergstrom, is that correct?

A. That's correct.

Q. Other than the crocidolite, are you aware of any other crocidolite or potential crocidolite exposure among the Conwed cases that you've reviewed?

A. There is crocidolite marked in at least five different places within the plant. There was piping or some component in the plant, so I really couldn't exclude some additional potential exposure. The people came up who tore up the pipe and one of these were were near by.

Q. Do you have any specific information in that record from crocidolite exposure from pipe covering?

A. Have I seen anything that says crocidolite in pipe covering in Conwed plant?

Q. Yes.

A. Yes.

Q. What have you seen in that regard?

A. How do we call -- USG sample survey?

Q. That's USG, you mean U.S. Gypsum?

A. Yes.

Q. These are asbestos -- an asbestos survey that USG has conducted after they purchased the plant from Conwed, is that what you're talking about?

A. Yes.

Q. That indicates that some samples of pipe covering contained crocidolite asbestos?

A. Yes.

Q. And the information that you saw -- first of all, did you actually see a survey or did you read in a deposition about this?

A. I saw four large -- I guess about four foot by two foot plans, detailed plans of the plant. And then a smaller plan of the plant. And then a five or six-page document which listed about 200 sites. So for example in the left-hand column there, there was a number running from -- the numbers ran from 1 to 200. In, say, I don't remember exactly, but the next column, there

was -- the location from which the sample was taken, and then the next column was the type of asbestos found, either chrysotile, crocidolite or amosite. There were about -- they went from, say, 1 to 132, consecutively, and then for some reason there were five or six missing. Then it went for another ten and then there were five or six missing. The number was virtually intact but there were some things missing and there were about 30 records where they didn't actually say crocidolite or amosite, it said positive or negative.

Q. Do you know what the date of the survey was?

A. I don't recall.

Q. Was there any information that you saw, either on the survey or anywhere else, which indicated when those materials were installed in the plant?

A. When they were installed?

Q. Right.

A. I don't recall.

Q. Was there any information, either in the survey or anywhere else, that you reviewed

that stated whether or not any of these six mesothelioma cases we've been talking about worked with pipes or pipe covering or had any exposure to those particular products that you mentioned?

A. Could you ask the first part of the question again.

(Record read)

A. Are you asking to my recollection if any of these guys worked with pipe covering?

Q. Right.

A. I believe so.

Q. Do you know who?

A. No.

Q. All right.

Q. Would the information you had in that regard be retained in the files we looked at earlier and are going to have copied here?

A. I believe so.

Q. Really what I'm getting at, I'm trying to find the sources of your information with respect to that question whether any of these six mesothelioma cases would have had exposure to crocidolite and pipe covering. You told us you got six case files -- five of them here other than

Mr. Bodway. Then you told us you have this USG survey. Is there any other data or information that you've seen --

A. Let's look at the list for a second.

MR. WILL: You're talking about the exposure of the individuals?

MR. BROWNSON: Right.

A. Mr. Bergstrom, I've looked at the three documents which are Bergstrom something. They make reference, I think in at least two documents, to the use of crocidolite.

Q. Let's stop there. With respect to whether Mr. Bergstrom was exposed to crocidolite, you have seen three documents. Are those the three which are listed on the first page of Exhibit 13 under Bergstrom?

A. Right.

Q. I guess maybe my question wasn't clear, maybe it was. What I am wondering is, have you seen any other information concerning exposure to pipe covering among these six cases?

A. I don't recall. I may have, I may not have.

MR. WILL: I have not given him

their depositions, but based on your line of questioning, maybe I should do that. He did read Manisto, excerpts from that.

Q. Now, do you have a copy of this USG survey that you were talking about?

A. Yes.

Q. And would your copy be the copy that you relied on for the statements you made here today for the USG survey?

A. Yes.

MR. BROWNSON: Can we get a copy of that?

MR. WILL: I think you have a copy of that.

MR. BROWNSON: I would like to see -- he said there were some pages missing.

MR. WILL: There aren't pages missing. If you look on the sample survey, it's missing numbers, not pages. They went around the plant and took samples out of each of the pipes and they put a number on them. The survey that he has lists samples 1 through 185. But there are some numbers

in the sequence -- there are some sample numbers in the sequence that are not reported. I can give you a copy of it.

Q. I'm just wondering, Mike Goldman asked, is that survey listed on your Exhibit 13?

A. I don't think so.

Q. It's not something we requested earlier. Just to follow-up. Mr. Will advises this is something you've seen in the last week?

A. Yes.

Q. And it came from Mr. Will?

A. Yes.

Q. And you have a copy in your possession?

A. Yes.

Q. So if we wanted to see the specific survey that you reviewed, you still have that copy?

A. I do.

Q. Now, have you been asked by Mr. Will or anybody else on behalf of Union Carbide to attempt to render any opinions in this case against Owens-Corning or any of the third-party defendants in this lawsuit?

A. No.

Q. Are you aware that there are third-party defendants in the lawsuit?

A. Yes.

Q. And whether or not you've been asked to render any opinions at the present time with respect to those third-party defendants, do you have any opinions as to whether any of those third-party defendants, based upon the material you have seen, have anything to do with any of these six mesothelioma cases?

A. No.

Q. By that, you mean you have no opinion?

A. I haven't reviewed any.

Q. Okay. Let's go back to Exhibit 13.

(Recess taken)

BY MR. BROWNSON:

Q. When we broke, we were about to look at Plaintiff's Exhibit 13, which is your list of documents requested at the last deposition. Before we do that, can you think of anything else that is not on this list Exhibit 13 that you've reviewed in connection with this case or your

opinions in this case?

A. Pertaining specifically to Calidria?

Q. Right.

A. Nothing comes to mind.

Q. Have you reviewed any of the deposition exhibits or documents from Dr. Kagan's deposition taken earlier this week?

A. No, I haven't.

Q. Let's take a look at Exhibit 13. What I want to do is go through the request that we had made for documents and see if I understand where these things fall on the list.

A. All right.

Q. And the first thing I want to look at, we had documents he maintained the in office at Bryn Mawr. There were some specific things. The first is the medical summaries of the six Conwed workers, and I've seen five and you have number six of Mr. Bodway which you'll provide to us.

The second thing, correspondence with Dr. Kevin Brown.

A. I checked my files and I didn't see anything -- I was mistaken, I didn't see anything

TABLE 7

FIBER LENGTH DISTRIBUTION—OPTICAL MICROSCOPY

Preparation	Dispersion Method	Study	Percentage longer than stated length (μm) 4	5	10	20	40
UICC B	Aerosol	Present		100	68.2	40.5	12.6
UICC B	Aerosol	Beckett[a]		100	32	11	5
UICC B	Aerosol	Timbrell[b]	100		22.3	11	1.7
UICC B	Alcohol	Timbrell[b]		100	46.9	21.5	7.0
UICC B	Celloidin	Timbrell[b]		100	53.6	16.9	3.8

FIBER LENGTH DISTRIBUTION—ELECTRON MICROSCOPY

Asbestos-dispersion method	Instrument	Study	Percentage longer than stated length (μm) 0.2	1	2	5	10	20
UICC B aerosol	TEM	Timbrell[b]	100	73.6	54.4	27.0	9.6	3.2
UICC B aerosol	SEM	Present	100	88.2	64.2	25.5	6.7	1.9
JEFFREY aerosol	SEM	Present	100	85.0	50.7	24.9	8.0	2.7
			5	10	20	30	50	60
UICC B aerosol	SEM	Beckett[a]	100	45	12	6		1
UICC B aerosol	SEM	Present	100	26.4	7.4	2.3	0.8	
JEFFREY aerosol	SEM	Present	100	32.1	10.8	5.5	2.7	

[a] From Figure 2a in Beckett, 1973.

[b] Modified from Tables 6, 7, or 8 in Timbrell, 1970a.

particles analyzed were greater than 10 μm in length and had a mean diameter of 0.42 μm. In the present study using aerosolized samples drawn directly upon Nucleopore filters and subsequently gold coated, it was found that 34.0% of the combined fibers and fiber clusters or 28.1% of all particles (fibers, fiber clusters, and nonfibrous particles) in the Coalinga preparation were greater than 10 μm in length and had a mean diameter of 2.92 μm. In a subsequent study by Siegrist and Wylie (1980), the Coalinga preparation (referred to as short-range chrysotile) was characterized for particle size distribution by both transmission and scanning electron microscopy. The samples were prepared by hand swirling in distilled water and dishwashing liquid (for SEM analysis) and by ultrasonification in water for 10 min (for TEM analysis). The results demonstrated that by both SEM and TEM more than 90–95% of the particles were less than 10 μm in length and more than 95% of the particles were less than 1 μm in diameter. In our study using aerosolized samples 71.9% of the total number of particles were less than 10 μm in length and 68% of the total number of particles were less than 1 μm in diameter.

The differences between these two methods of sample preparation and analysis

comments or his comments with respect to those?

A. Possibly.

Q. And are there any particular cases that you can recall where -- that you have discussed with Dr. Kevin Brown where the issue was, was this a pure chrysotile mesothelioma or was it something else?

A. I don't recall specific things.

Q. Just backing up a minute, do you know if that was a question in that esophageal cancer case you mentioned earlier with Union Carbide, the question being whether there was exposure to something other than Calidria, another kind of asbestos, other than Calidria?

A. I don't remember the case.

Q. Whether there was a question or not, you just don't recall?

A. I don't remember.

Q. Do you maintain any file with respect to that case?

A. I believe so.

Q. And do you maintain any file with respect to those other two cases you worked on that dealt with Calidria exposure?

A. I believe so.

Q. And to the extent those files still exist, would they be at your office in Bryn Mawr?

A. Yes.

Q. Do you know if you reviewed any tissue or tissue slides in that esophageal cancer case?

A. I don't believe so.

Q. Do you know if going back to Exhibit 13 in the correspondence with Dr. Kevin Brown, do you know if that correspondence dealt in any way with the question of -- or any questions dealing with the Yeager and Langer paper about the exposure or the test performed with macrophages?

A. I don't recall, it's possible.

Q. And the reason I bring that up, we'll get to it in a minute. One of the documents was a critique of that paper by Dr. Addison. Do you recall that?

A. Yes.

Q. Are you aware of any similar critiquing with respect to that paper done -- which would be reflected in your correspondence with Dr. Kevin Brown?

A. Formal critique --

Q. But any critique?

A. Critical comments?

Q. Yes.

A. I don't recall. Perhaps.

Q. All right.

Q. You were about to say something. Did something pop in your mind on that question?

A. Indigestion.

Q. Are you aware of any other written critique, whether formal or informal, of that particular paper, the Yeager and Langer paper other than the one you provided to us by Dr. Addison?

A. No.

Q. The next thing we requested was the category of documents, a file maintained in Bryn Mawr, Conwed hygiene data. Did you find anything that fell under that category?

A. Yes.

Q. Where would that be reflected in Exhibit 13?

A. I don't think I realized that you wanted that to be listed so it's probably not

Ilgren
A. No, sir.
Q. Are you aware of such modeling he has done with respect to other types of chrysotile fiber?
A. Yes.
Q. Has that been published work?
A. Yes.
Q. Is there also unpublished work?
A. I assume.
Q. Is there any that you have seen?
A. No, sir.
Q. So what you have seen is his published work concerning modeling of chrysotile degradation or dissolution?
A. Yes.
Q. Do you recall offhand which chrysotile he used? First of all, was it all Canadian?
A. I don't recall whether it was Canadian or Rhodesian. I don't recall.
Q. That's kind of a small point, but let's put it this way, do you know if he has done -- when I earlier asked you whether he had done such modeling with respect to Coalinga, I wasn't limiting myself to Union Carbide Coalinga,

0072

Ilgren
I was limiting myself to the deposit.
A. Yeah, sure. Yes.
Q. So basically if I can summarize the request you have made of Rimstidt, it's that you would like him to model the dissolution or degradation of Coalinga type chrysotile and compare it with other types that he has modeled?
A. Yes, sir.
Q. Is that a request that you have made of Union Carbide, to do that work?
A. Not yet.
Q. Is that a request that you intend to make?
A. Yes, sir.
Q. Let me ask you the same question. Do you have any estimate of how long that work would take, if you get the go-ahead to do it?
A. No, sir.
Q. Have we now covered all of the original topic, laboratory work that you have requested with respect to Coalinga asbestos? Have we now covered it all?
A. Off the top of my head, yes.
Q. Have you requested any TEM analysis of

0073

Ilgren
the fiber itself?

establish what's what and what's where.

A. I think that's why it's not on the list. We discussed this and he said he would provide you with things which -- he would not provide you things that you thought you already have.

Q. So with respect to the category of Conwed hygiene data, what you have is three things, the in-test report, the Union Carbide air testing, and then this portion of this report from Dr. Gaffney?

A. I believe so.

Q. Is there anything else that you have considered in your mind to be high general testing data or exposure-type data from Conwed?

A. Not to my knowledge.

Q. The next thing, Cloquet's answers to Trevor, he didn't provide because we have it. You reviewed certain certain interrogatory answers from Conwed?

A. Yes.

Q. Have those included interrogatory answers in this case?

MR. WILL: I don't know if he knows.

dealing with Calidria fibre, but they're listed on the list?

A. Exactly.

Q. Were those some of these things -- I should address this to Trevor.

MR. BROWNSON: Is it your position we can get it as well as you can, so we should get it ourselves?

MR. WILL: That's part of it. The things that I told you that he paid for maybe are a little different category.

Q. Let's do this. I'm trying to move this along. Exhibit 13 is a list of everything that you found responsive to the document request from the first deposition?

A. That's correct.

Q. And all this stuff on Exhibit 13, however, has not been copied and provided to us, is that the case?

A. That's correct.

Q. As I understand it, certain things were not copied and provided because we had it already in the opinion of Trevor Will?

A. Correct.

University."

A. Page 2, top of the page Butler, 1980.

Q. That's a PhD thesis by Butler?

A. That's correct.

Q. All right.

A. All of the entry documents on the bottom of page 2 and top of page 3..

Q. That would be eight documents?

A. That's correct. Yes, eight documents.

Q. Now anything else other than those things?

A. I don't believe so, no.

Q. Let's just go through these things --

MR. WILL: There are nine of them.

THE WITNESS: Nine entries.

MR. BROWNSON: Nine entries, I'm sorry.

MR. WILL: Also we didn't send you a copy of a medical screening, we talk about the fact you that you had that.

MR. BROWNSON: What I'm wondering is there are certain documents that are reports Dr. Ilgren has that you didn't

provide because Dr. Elgren being -- you take the position that Dr. Elgren took some time and expense to get these. If he just gives them to us we'll get the benefit of that time and expense without paying for it.

MR. WILL: Right. If you care to ask him, he can tell you how to get them yourself. They're available.

MR. GERSON: You reviewed this in connection with this case and you're taking the position that you won't turn them over.

MR. WILL: I'm not saying we won't them turn over. We're not going to give you the benefit of the cost that he incurred.

MR. BROWNSON: Let me -- whether or not that's -- let me ask some questions about it.

(Recess taken)

BY MR. BROWNSON:

Q. I wanted to ask you about these items on Exhibit 13 that we've highlighted under the category of identified but not provided because of

cost. And the first one is this Butler PhD thesis from the University of Cardiff, Wales in Britain.

A. That's right.

Q. Is that something that you relied on in any way for any of your opinions in this case?

A. I would have gotten the document, yes.

Q. Do you know if any of the serpentine rocks and minerals discussed in that PhD thesis came from California?

A. They were Coalinga samples mentioned.

Q. Do you know if they were specified as coming from the Union Carbide mine or just Coalinga samples?

A. I just can't recall, I'm not sure.

Q. For the record, I think we're entitled to get this stuff. But after stating that, what expense would you view as reasonable to compensate Dr. Ilgren for his trouble?

MR. WILL: Can you give me two minutes to go talk with the doctor for a second about this?

Q. You also identified, Dr. Ilgren, these nine entries which fall under the category

of things you got and are responsive but you don't want to produce because of this cost. Did you rely on any of those nine reports in reaching any conclusions or opinions which pertaining to this case?

A. I have to recheck it. I believe so, but I have to recheck the details.

Q. The final thing is the Pinkerton thesis in 1982. Is that something you relied on in any way for opinions and conclusions on this case?

A. Yes.

Q. And I just had a discussion with Trevor Will in the hallway and agreed at the end of the deposition we'll discuss with him making a request for this stuff, but we still do request it. We'll discuss what arrangements can be made, if any, to get that.

Q. Let me ask you this. In your published book, Mesothelioma of Animals, that we were talking at the last session of the deposition?

A. Right.

Q. Do you compile or list any of the

And again, are those things you looked for and listed on Exhibit 13?

A. Right.

Q. All right.

A. The only thing I couldn't find was this J. Haart, Jean Haart. I was mistaken, I didn't have it, I thought I had it. But I didn't.

Q. What's listed as Haart, J., which is Jean Haart?

A. Yes.

Q. Correspondence?

A. Yes.

Q. You couldn't find that?

A. Correct.

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Q. And on my letter I talked about documents or correspondence from Dr. Haart, is that the same person?

A. I think that's the same person.

Q. Now let me ask you this question. With respect to these published and unpublished documents or reports concerning -- that we requested at the deposition, did you limit your review to those which specifically deal with Calidria asbestos?

A. I think there are some others here in fact that deal with JM Coalinga, so the answer is no.

Q. Earlier you had made the distinction between Coalinga and the other Coalinga fibers, and I'm wondering if your review here was reviewed to just Coalinga or was a broad category of --

A. I would say there are a number where I couldn't categorize, like it was a Union Carbide product or something else.

Q. Then the other thing we listed was NAAC, which is an acronym, letters to Conwed, is that shown on Exhibit 13?

A. It's third from the bottom, page 1, Bergstrom, 21, October 1960, letter from Algerd E. Jerome.

Q. Next is correspondence from NIOSH, another acronym of Dr. -- Drs. Jean Haart and Charles Lorberau. I think it's NIOSH. I did see some documents in that regard. So you have produced some documents. Are there any of those documents that you found but did not produce?

A. No, not to my knowledge.

Q. The other things were documents that

are not on file at Bryn Mawr. The first thing is the Oxford calendar. Were you able the find that?

A. Yes.

Q. One of the things you produced, I noticed, there was some photocopy pages from something about -- was it green collegiate Oxford?

A. Yes.

Q. What was that?

A. That's the Oxford calendar.

Q. Then the final thing was the interim or final report on this Shubik study on the health and safety executive interim report, Oxford calendar listing of Professor P. Shubik.

Q. Was that something that was provided to us?

A. Yes.

Q. Was that something that was provided to us?

A. Yes.

MR. GERSON: Now I want to look at a few of these documents in a little more detail. The first thing I got, why don't we mark this as Exhibit 14.

(Whereupon, letter dated February

16, 1994 marked Plaintiff's Exhibit 14 for identification, as of this date.)

Q. Exhibit 14 is one of the documents you produced after your last deposition. This is a letter to you from John Addison.

A. Right.

Q. What's the date?

A. 16 February, 1994.

Q. I'm looking at the first page, which is the message to you from Dr. Addison, and he makes reference to transcript discussions. What transcript are we discussing here?

A. A portion of Art Langer's deposition.

Q. Do you know -- was Dr. Art Langer's entire deposition sent to Dr. Addison or were some portions sent to Dr. Addison?

A. A portion.

Q. What portion was sent to --

A. It could be the first line at the top -- I believe -- "In a given amount of amosite would you expect to find more or less or the same number of fibers as an equivalent weight of Calidria."

Q. All right. So what you sent to Dr.

Addison was the portion of the transcript where that question was asked?

A. Yes. It was more than this. I think a whole page. But I thought I had put it in. But maybe I didn't put it in.

Q. What you sent to Dr. Addison was a page from the transcript that contained one question and an answer or were there other questions and answers?

A. I think there were three or four questions and answers.

Q. And were all of those questions and answers on this topic of comparing Calidria to amosite in terms of mass or number of fibers?

A. Yes.

Q. And whose idea was that to send that to Dr. Addison for his comments?

A. Mine.

Q. And why was it that you wanted Dr. Addison's comments on those questions and answers?

A. I didn't understand what Art was talking about.

Q. And Dr. Addison's letter says, there is not much to say about the transcript

discussions except that they seem to be fairly uninformed about the nature of asbestos. Is he referring there to the question or the answer?

A. Both.

Q. Then he says, "I have put a few ideas down on paper for you." And what I'm wondering is where do his ideas start here? Is that on the third page of this exhibit?

A. In fact, I believe the entire thing -- the entire document, these three pages are written by Addison. The Q and A page that I sent to him is not here.

Q. So what we got in this Exhibit 14 is we got the first page is a cover letter from Dr. Addison. Then there are three pages of Dr. Addison's commentary.

A. Right.

Q. And Dr. Addison's commentary is on -- is with respect to Dr. Langer's description of this comparison between amosite and Calidria fibers, is that fair to say?

A. Right.

Q. Now toward the bottom of the second page of the exhibit, which is Dr. Addison's

commentary, he states -- I'm reading from down here. He states --

A. All right.

Q. -- "If something is to be made of the differences in size of the different asbestos varieties, the most important thing to do is to compare fibre populations." Do you see that?

A. Yes.

Q. He goes on to say, "The best fibre populations to compare are the airborne fibers since they are the one that constitute the hazard and create the risk."

A. I see it.

Q. Is that a statement you agree with?

MR. WILL: Which part of it?

MR. BROWNSON: That the best fibre populations to compare are the airborne fibers since they're the ones that constitute the hazard and create the risk.

MR. WILL: Are you asking him to agree with the statement that the airborne fibers constitute the hazard and create the risk? Are you asking him to agree to the part of the statement that the best fibre

populations to compare are the airborne ones or are you asking him to agree with both?

MR. BROWNSON: I'm asking whether he agrees with the statement as written by Dr. Addison, whatever its implications may be.

A. Well, I would like to know what the airborne fibers look like. I would like to know if there's any fibre information from what you see in the lung, of course, when you dig it out of the ground.

Q. Is Dr. Addison saying here, as you understand it, that in terms of looking at the fibre populations, what is important to him is what's in an aerosol as opposed to what's in the ground or in a pellet or something else?

A. I believe so, yes.

Q. Do you agree with the general proposition that in a given unit mass of fibre, there are more Calidria and amosite fibers simply because they're smaller?

A. Yes.

Q. Now, have you received any other -- or is there any other communication or

correspondence or letters or reports or documents from Dr. Addison dealing with this case?

A. Not that I recall.

Q. So as far as his involvement in this case such as it is, you sent him this page of pages of Dr. Langer's deposition that you had questions on and he returned Exhibit 14 as his comments?

A. Yes.

Q. And other than that, there's nothing in writing from Dr. Addison with respect to this case that you're aware of?

A. Aside from comments on Yeager --

Q. Let's go to that next.

(Whereupon, Dr. Addison's comments document marked Plaintiff's Exhibit 15 for identification, "as of this date.)

Q. Exhibit 15 are comments from Dr. Addison dealing with this paper by Yeager and other authors, is that correct?

A. Yes.

Q. Is this exhibit something that Dr. Addison typed up and sent to you?

A. Yes.

Q. What was the request that you presented to Dr. Addison that resulted in this Exhibit 15?

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Q. What question or request did you pose to Dr. Addison that resulted in this exhibit, what prompted him to write this, what was the question?

A. Principally, more information about the fibre types and the populations and the lengths and widths because he's used all these different fibre types.

Q. Now first of all, you're familiar with this paper that is being critiqued, the Yeager paper.

A. I haven't looked at it in a while but I'm familiar with it.

Q. Are you aware of any study other than the study reported in this paper where human cells of any type, whether macrophages or other types of cells, were dosed with Calidria asbestos to see what the effects were?

A. I think it's Russo et al., which I should have put on here.

Q. All right.

A. 1988. Yes, I put it on here. It's

on page 7, Russo, et al.

Q. So that's page 7 of Exhibit 13, your list of papers?

A. Yes.

Q. You list a paper by Russo, et al.

Q. And as you understand it, what he did was took some macrophages in vitro and dosed them with asbestos?

A. It's an extension of Yeager, et al., 1983.

Q. Do you know what types of asbestos were used in that paper?

A. I recall it was Coalinga and something else.

Q. By Coalinga, do you know if it was Calidria or not?

A. I don't recall. I think it was RG-144.

Q. Did you ask Dr. Addison for any critique of that paper as well or just of the Yeager paper?

A. Just Yeager.

Q. Other than this paper by Russo 1988, are you aware of any other experiments where human

cells were dosed with Calidria asbestos to see what the results would be?

A. Langer et al. 1988.

I'm sorry, 1978.

Q. Any others?

A. This is in vitro --

Q. I'm not talking about just published papers, I'm talking -- the question was experiments.

A. Nothing immediately springs to mind.

Q. Have you suggested to Union Carbide or Union Carbide representatives, there ought to be experiments of this nature to see what the results would be?

MR. GOLDMAN: Can you just characterize "this nature."

Q. Dosing human cells with Calidria asbestos?

A. No.

Q. Have you suggested any animal experiments to Union Carbide or Union Carbide representatives whether they are inhaling or injecting or whatever with respect -- using Calidria asbestos?

Addison was the portion of the transcript where that question was asked?

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Q. Dosing human cells with Calidria asbestos?

A. No.

Q. Have you suggested any animal experiments to Union Carbide or Union Carbide representatives whether they are inhaling or injecting or whatever with respect -- using Calidria asbestos?

A. As mentioned in my first deposition, only the recharacterization of Muhle dusting cloud, I haven't actually said we should dust animals.

Q. Do you know when it was that you requested of Dr. Addison that he review the Yeager paper?

A. The date?

Q. Well, not the exact date, when was it that you made that request?

A. I don't recall.

Q. Do you know -- was it in connection with the deposition that Dr. Langer gave in this case or was it independent of that?

A. I don't recall.

Q. Is there anything in the comments of Dr. Addison in Exhibit 15 that you specifically disagree with or -- let's cover that question --

MR. BROWNSON: Let's break now. I have a series of questions. Maybe it will help to move it along if you look it over at lunch.

(Lunch recess: 12:30 p.m.)

Afternoon Session

1:30 p.m.

E D W A R D B. I L G R E N, previously sworn, resumed:

(Record read)

MR. BROWNSON: I'll pose the question again.

MR. WILL: I object to the form. The document is four or five pages long and it's hopelessly vague and multiple questions.

Anyway go ahead.

A. Anything specific?

Q. Right.

A. You would like to point to -- point to a sentence?

Q. I'm going to go through it. But before I did that, I want to ask the general question, is there anything you wanted to point out to that you can disagree with?

A. General answer, I disagree with list conclusions.

Q. Let me point you to the last sentence of the text, which reads -- should we see -- do

you have a copy of that? Specifically I'm pointing to the last sentence of text that reads as follows: "The fact that large numbers (5 times 10 to the ninth) of fine Calidria chrysotile fibers were cytotoxic to human macrophages is scarcely surprising and the fact then that even larger numbers (1.15 times 10 to the eleventh) were more toxic in even less surprising."

Q. Do you agree with that statement?

A. Yes.

Q. Now, why was it that you asked Dr. Addison to comment on the Yeager, et al. paper?

A. I had questions about the validity of comparing certain Calidria fibers about UICC fibers.

Q. What I'm wondering, why did you go to Dr. Addison as opposed to someone else for answers to those questions?

A. Such as who?

Q. Why was it that you chose Dr. Addison to ask the questions to as opposed to someone else?

A. He's a great world authority.

Q. What is the area in which Dr. Addison

is a world authority?

A. The quantitative assessment and qualitative assessment of mineral fibers.

Q. And do you know if Dr. Addison has done any of his own characterization of Calidria asbestos fibre?

A. I believe he has.

Q. And do you know if that work has been published anywhere?

A. I believe it hasn't.

Q. But despite the fact that Dr. Addison has not published his work, you still rely upon him as a leading authority in the end, in the area of characterization of Calidria asbestos fibre?

MR. GOLDMAN: Object to form. I believe the response is -- as to what was unpublished was only one particular piece of work, not his work in general.

MR. BROWNSON: I understand that.

A. Yes.

Q. The answer is yes?

A. Yes.

Q. Now in addition to the comments by Dr. Addison which are contained in this Exhibit

15, did Dr. Addison provide you with any other written material or comments on the Yeager paper?

A. No.

Q. Now the copy I have of Exhibit 15 doesn't have table 1, it just tables 2 and 3. Do you know where table 1 is?

A. I can't find it. Mr. Will made me aware that table 1 wasn't there and there were two copies of tables 2 and 3. I need to find that. I searched for it and if I don't find it, I'll call Dr. Addison.

Q. There's a table 1?

A. As far as I recall. I'm not sure. He obviously mentions table 1 in the text.

Q. Can you get a copy of that and provide it to us?

A. Yes.

MR. GOLDMAN: He can try.

Q. Now did you have to pay Dr. Addison to do this review and write this report?

A. Yes.

Q. And how much did he charge for that work?

A. Three hours at $300 an hour.

Q. And is that something you paid personally or did you pass that bill on to Union Carbide?

A. Initially paid it myself and then passed it on.

Q. So Union Carbide reimbursed you for that?

A. Yes.

Q. That charge by Dr. Addison?

A. Yes.

Q. In addition to the charge for Dr. Addison, have you incurred other outside charges, by that I mean charges from other people in connection with your work with Union Carbide in this case, something other than charges for your own time?

A. Yes.

Q. Can you tell us what those are?

A. I think in acquiring the IITRI documents, I paid Jean Graf for the time and expenses incurred in getting those materials.

Q. And again that charge was something that came in an invoice from her and was ultimately paid by Union Carbide?

A. I believe so.

Q. Now can you give us an exact figure if you can, an estimate of how much you have charged Union Carbide all together for your work on this case?

A. An exact figure?

Q. If you have an exact figure. If you don't, can you give us a ballpark?

MR. WILL: I object. Go ahead and answer, on relevance.

Q. All right.

A. Let's say I've put over 1,000 hours.

Q. And what is your charge that you charge for your work in this case?

A. $200 an hour.

Q. And of the $1,000 plus hours put into this case, how much of that has been in the last year, if you can give me an estimate?

A. I don't know. It's mainly over the last two years.

Q. The last two years?

A. Yes.

Q. Over that period of time, let's take the last two years, what portion of your time, to

use a lawyer's term, billable time, have you spent working on this case?

A. And Coalinga-related matters?

Q. Let me ask you if you break it down just for this case.

A. I can't.

Q. Well, then, let me ask some more general question of Coalinga-related matters, which I understand would include this case?

A. All right..

Q. What's the question?

A. The question.

Q. The question is what's the percentage of your billable time or billings for the last two-year period that have been devoted to these Coalinga-related matters that include this case?

MR. GOLDMAN: Let me object to form. Bob, this is a good time to clarify this point. The only asbestos fibre sold by Union Carbide is what Union Carbide mined in King City. You know from our interrogatory responses and Dr. Ilgren has been retained by us as a scientific medical expert on matters pertaining to what Union

Carbide mined, that same source. If you're going to use as general terms as pertaining or relating to, I mean it was clear as we've said in our interrogatory responses, all of Dr. Ilgren's consulting for Union Carbide pertains to Calidria and Calidria -- all of Calidria comes from Coalinga and so there are -- all of his time in some way would have to be related to that.

MR. BROWNSON: Maybe my question wasn't clear. I wasn't implying otherwise. I understood that.

Q. I'm not asking at this point other work you might have done for Union Carbide, for radiation or something. I'm just asking for work you've done for Union Carbide with respect to Calidria or Coalinga asbestos.

A. And the question is?

Q. My question is, what percentage of your billable time has that constituted during this past two years since you've been doing this?

MR. GOLDMAN: All the billable time for all matters --

practice or has your time been spent on these different projects?

A. Different projects.

Q. I wanted to ask you when we were talking about the charge from John Addison --

A. Yes.

Q. -- I forgot to ask you earlier with respect to Exhibit 14.

A. Yes.

Q. Which was his comments on the Langer deposition.

A. Yes.

Q. Was that included in that $900 or was that charged separately?

A. I don't recall. I think it was included, but I don't recall.

(Whereupon, letter dated January 5, 1994 marked Plaintiff's Exhibit 16 for identification, as of this date.)

Q. We're now looking at Exhibit 16, which is one of the documents you produced since the time of your last deposition. This appears to be a letter from you to Dr. Charles Lorberau of January 5, 1994.

A. No.

Q. Was there anyone who told you that they were using the samples to -- for any animal experiments or had used them for any animal experiments?

A. No.

Q. Was there anyone on the list who told you that they were using the samples in connection with any lawsuits arising out of exposure to Calidria asbestos?

A. No.

Q. As best you can recall, what were the purposes that were given to you by these people for wanting these Calidria samples?

A. Analytical.

Q. And why was it as related to you that these people wanted to analyze these samples?

A. I don't understand what your question is.

Q. I assume -- did any of these people tell you why they wanted to analyze these samples?

A. I mean, as I recall, one, Brian Davis was interested in the x-ray spectra.

Q. Any other --

A. Another, Verma -- Davis's paper is on page 5 of the Exhibit 13.

The Ontario group on page 2, Nancy Clark of the Health Sciences Center at McMaster had been doing some infrared-type of analyses. That work is mentioned on Exhibit 13, page 4, under Verma, D. She's the head of that department. Verma is the head of that department.

Q. Reference in page 4, it says IR curves.

A. Infrared.

Q. Does that complete your answer then?

A. Infrared?

Q. Do you know what the reason was that anyone other people on this list obtained these Calidria samples?

A. No.

Q. Have you found out or been told what any of these other people were doing with the Calidria samples?

A. No.

Q. Did you receive a response from Sergeant Mike Wantland, Occupation and Analytical Department at Brooks Air Force Base in Texas.

What was he doing?

A. Nothing.

Q. Do you know why he requested these samples?

A. It was just a standard that they wanted to have as a reference standard. When I spoke to him, as I recall, he wasn't doing anything with it.

Q. Do you know if his work at Brooks Air Force Base had anything to do with asbestos sampling or analysis in terms of asbestos-contained materials in the buildings at the air force base?

A. It might have, I'm not sure.

Q. How about C.J. Martin at Oxford University, did you talk to that person?

A. Yes.

Q. And is this someone you knew from Oxford or is this a new name to you?

A. It was a new name. He's not at the university. He's at what they call at the open university, which is a different outfit up on the hill.

Q. And what did you learn from him when

Q. Have you ever done any work with Richard Wilson?

A. No.

Q. Do you know him at all?

A. I know of him. I've seen him. I've heard him speak.

(Whereupon, document headed "fibre Size Determination and Distribution of Calidria Chrysotile Asbestos by Transmission Electron Microscopy" marked Plaintiff's Exhibit 18 for identification, as of this date.)

Q. Now we're looking at Exhibit 18, which is another document that you produced to us following the last deposition, and it's entitled "fibre Size Determination and Distribution of Calidria Chrysotile Asbestos by Transmission Electron Microscope."

Have you had a chance to read this paper?

A. Very quickly.

Q. When did you receive this?

A. I don't recall.

Q. All right.

A. No.

MR. BROWNSON: Then Trevor Will has something on you there, because he has.

Q. But in any event, you've had telephone discussions with Mr. Meyer's?

A. Yes.

Q. Have you had any telephone discussions with anyone else from KCAC?

A. No.

Q. Have you had any correspondence with Mr. Meyers?

A. Yes.

Q. Is that substantive correspondence or enclosed as a paper or send me a paper sort of thing?

A. I don't recall? I think it's more substantive.

MR. GOLDMAN: John Meyers is retired from KCAC Inc.

Q. Let me ask you this. I thought I asked you this. Have you had conversations with anyone from KCAC other than John Meyers?

A. Not that I recall.

Q. This particular document, Exhibit 18,

came from Mr. Meyers?

A. I believe so. I don't recall.

Q. What was the request that you made that resulted in this document? In other words, did you say, give me any documents you have, size distribution, or was it a more general question?

A. I believe it was size distribution.

Q. Why were you interested in seeing what he had on that issue?

A. I'm interested in any data pertaining to Calidria.

Q. Have you received any data from KCAC other than this Exhibit 18 and then the other one, actually there's a second one which we'll get to in a minute which is a similar paper with a different date. You said you received those two things from him. Did you receive anything else?

A. I said I wasn't sure whether I received these. I may have received them. I don't believe I received anything else from KCAC.

MR. BROWNSON: Let's mark this other one.

(Whereupon, document headed "fibre Size Determination and Distribution of

RG-244 Calidria Chrysotile Asbetos by Transmission Electron Microscopy" marked Plaintiff's Exhibit 19 for identification, as of this date.)

Q. Exhibit 19 is a second fibre size distribution paper done for KCAC Inc. Except this one is dated February 3, 1993, and Exhibit 18 was dated March 30, 1991.

A. Yes.

Q. And as a general matter, it looks to me what Exhibit 18 is is a TEM size distribution analysis of four different sample types of Calidria asbestos, wherein 19 they just did RG-244?

A. I believe so.

Q. Has Mr. Meyers or anyone else communicated to you why they -- this follow-up TEM size distribution analysis was done on the RG-244?

A. No.

Q. Can you tell from your review of the papers why the follow-up was done on the 244?

A. I haven't read this in a while so I couldn't say at this time.

MR. WILL: I believe you misspoke.

analyses with TEM.

Q. All right.

A. I think that's it.

Q. You're looking at Exhibit 13, which is your list here. With respect to this analysis by Naumann, there are four entries by Naumann on your paper. Is it one of these that you think is this TEM analysis?

A. Yes. I don't think it was solely a TEM analysis. I'd have to go back and check. I think it contains some TEM data.

Q. Do you know if it contains any size distribution data?

A. I believe it contains internal and external diameter, fibril diameter data. I think Naumann and Dresher, 1966.

Q. How about fibre and language, is that data contained in there?

A. I think it's just diameter.

Q. How about the series of articles on Exhibit 13, how about the series of Yada, you mentioned some of that may be TEM size distribution?

A. Yes.

Q. Can you refer me to one or more of those that might contain that information?

A. I think it's Yada 1979. Not 100 percent positive, but I think it's the 1979 paper.

Q. Have you --

A. There was also -- I believe some EM data -- I don't quite recall, he had duplicated Naumann. I seem to recall EM data and I seem to recall Chwastiak.

Q. On this Chwastiak reference on page two, Exhibit 13, it doesn't indicate a source.

A. I'm sorry, that's a UCC document.

Q. That's one of the things you produced in the documents?

A. -- Yes.

A. It's one of the documents I assume you would get from Kelley Drye.

Q. It's not one you got from your box?

MR. WILL: It was in there.

THE WITNESS: I don't think so. I don't know.

MR. WILL: I think it was in there.

Q. In any event, you got it from Union Carbide or their lawyers, Kelley Drye & warren?

A. Yes.

MR. WILL: Off the record.

(Discussion off the record)

MR. BROWNSON: On the record.

Q. On Exhibit 18, I would like to refer you to page number TEM-3, in paragraph number 4 at the bottom of that page reads, "The breathing of asbestos dust may cause serious bodily harm and we can make no warranty or guarantee that the results contained herein indicate that safe levels of exposure exist." Do you see that?

A. Yes.

Q. I take it that with respect to the Calidria asbestos that they're analyzing in this Exhibit 18, you disagree with that statement?

A. It's dust. People do take precautions for high levels of dust, such as nuisance dust.

Q. Let me ask you this. It's your opinion, I understand, that Calidria asbestos dust in any concentration cannot cause mesothelioma?

A. That's correct.

Q. Is it also your contention that Calidria asbestos dust in any concentration cannot

find was 1.03 FML. My understanding, for example, the types of activities at the Conwed plant that would have produced high levels, or the highest levels would be, say, blow-downs as opposed to finish or sanding.

I attempted to, say, reconstruct the type of cumulative fibre-year dose a Conwed workman would have incurred in a more or less worst case scenario, and in that context I would have estimated that a man in the plant in a so-called blow-down setting would not have worked there in that kind of situation for more than six hours a week. And that would have gone on for 50 weeks a year, which would have been 300 hours a year. If one takes 2,000 hours as the standard work year and divides 300 by 2,000, one ends up with 0.15 years.

If one then takes as the reasonable estimate of the kind of upper working limit, maximum working limit that one might find in a ceiling tile plant for Calidria as one being the highest level that they found, the most one could incur in terms of a fibre year dose in a year would be 0.15 years. The generally recognized

threshold figure for clinically manifested is 50 fibre years which is generally placed on the Rochedale Textile Work as cited by Doll & Peto into their 1985 asbestos document.

Q. All right.

A. If one then asks the question how many years that Conwed workman would have to work in order to achieve a 50-fibre year cumulative so-called threshold dose, one would then I believe need to divide 50 by 0.15. But the threshold dose of 50 fibre years is truly predicated on a textile milling situation in which there is not, say, 4 percent or 8 percent -- where 5 micro fibers, but there would be 30 or 40 or 50 percent long fibers. So what I would do is I would take the so-called 300 years and multiply it by at least two.

Q. Where do you get 300 years?

A. That's 50 fibre years divided by 0.15 years.

Q. All right.

A. That's 333, I think, something like that.

Q. So your bottom line is that a worker in the Conwed plant would need 666 fibre years?

A. My bottom line is that an individual would need to work for 666 continuous years in order to achieve sufficient exposure, in theory to get Calidria-related asbestosis.

Q. How do you define clinical asbestosis --

A. A S T.

Q. That is 1/1 --

A. Whatever -- you know the standard reference. The 1986 document in the American Review of Respiratory Disease.

Q. As a result of these calculations you made, you believe a worker would have to work 666 years in the Conwed plant to get clinical asbestosis?

A. From Calidria chrysotile exposure.

Q. How do you explain the fact that many workers do have clinical asbestosis after working in that plant?

MR. WILL: I object to the question. It assumes a fact that is not in evidence. Do you want him to assume for the sake --

MR. BROWNSON: I want him to answer the question if he can.

A. I want you to ask the question again.

Q. I'll ask this, are you saying there's no worker in that plant that has clinical asbestosis, no worker has come out of that plant with clinical asbestosis?

A. I don't know what the MDA documents documents say with precise instance of disease.

Q. Are you saying there is no worker who has come out of that plant with clinical asbestosis?

A. I don't know.

Q. Have you reviewed the case of Mr. Eugene Smith?

A. No.

Q. Have you ever reviewed any of the asbestosis cases?

A. No.

Q. We spoke earlier when we started this answer, your opinion on asbestosis from a Calidria asbestos basis based upon a 79 HSE document. And I assume that HSE is Health Safety Executive?

A. Yes.

Q. Which is a British government group?

A. Yes.

Q. As I understand it, the HSE published a document dealing with a tile plant in Oxbridge.

A. Yes.

Q. Do you know where that was published?

A. What was published?

Q. That document.

A. It comes out of HMSO, Her Majesty's stationer's office in London.

Q. Do you have a copy of that?

A. Yes.

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Q. What do you know about the tile plant in Uxbridge? Was that a plant where ceiling tile was manufactured?

A. I believe so.

Q. And do you know when the ceiling tile was manufactured in that plant?

A. I believe from 1947 to 1981.

Q. And is it your understanding that amosite asbestos was a component in that ceiling tile?

A. It is almost pure amosite.

Q. The tile was made of almost pure amosite?

A. I believe so.

Q. And does that document contain asbestos air level measurements in the plant during production of that tile?

A. I believe so, yes.

Q. Do you know what those are?

A. What --

Q. The air level measurements of asbestos during production of tile in that plant?

A. Yes.

Q. What are they?

A. They're categorized I think by sanding, sawing, finishing, measurements in the office, measurements in the plant floor. I can't recall exactly every specific data set, but the high readings, as I recall, were from 6 to 10.

Q. Fibers per cc?

A. Yes.

Q. All right.

A. There was some activity that said it was greater than 20, but the highest reading that was categorize, as far as I can recall, was 6 to 10.

Q. All right. And in taking that data and trying to translate it to the environment

within the Conwed plant when Conwed was producing tile, was using Calidria asbestos, what you've done is used the data from the 1972 Union Carbide air monitoring survey to get your vessels in the Conwed plant?

A. Yes, in conjunction with these other data.

Q. And of course if a particular worker had an exposure greater than 1.03 fibers per cc, he would need less than 666 years of work in the plant to develop clinical asbestosis?

A. Yes.

Q. So are you saying a person couldn't get asbestosis from exposure to Calidria asbestos because it's dose-dependent, and in your view a very large dose would be needed?

A. Yes.

Q. You have attempted to calculate that dose by -- based upon the data from the Oxbridge plant as reported in the HSE 1979 report?

A. And the Conwed plant.

Q. Well, maybe we're arguing about semantics. I understand you're attempting to translate the English data or superimpose it to

the Conwed plant experience?

A. To some extent, yes.

Q. As I understand your opinion, you're predicating your opinion upon the fact that you believe none of the workers in the Conwed plant have clinical asbestos, is that right?

A. No.

Q. So there may or may not be some clinical asbestos, you just don't know?

A. There may be, there may not be, I don't know.

Q. What this opinion was that you just gave us is an opinion which is a calculation based upon data from the Oxbridge ceiling tile plant in England, transferred over to what you understand about the exposure levels in the Conwed plant, would that be fair to say?

A. To some extent.

Q. And with respect to data concerning exposure levels in the Conwed plant, one piece of data you have is the 1972 Union Carbide air level survey, correct?

A. Yes.

Q. And what other pieces of data do you

have concerning exposures in the Conwed plant?

A. As I recall, there are only three. The UCC 1972 document, the in-test document, and one other which I can't remember.

Q. All right.

MR. WILL: The Gaffney.

A. The Gaffney report.

Q. The in-test document, you understand that to be a test done in connection with some work on pipe covering in the boiler room of the plant, or the steam plant?

A. I believe so.

Q. With respect to trying to calculate the exposure levels of what you call a worst case scenario, did Conwed have a plan in doing blow-downs with the air hose?

A. Yes.

Q. Did you calculate or assume some value for those exposure levels?

A. Yes.

Q. What was that?

A. One.

Q. One fibre per cc?

A. Yes.

Q. So what you were doing when you made this calculation was you were assuming that a man doing blow-downs would be exposed to one fibre per cc during his blow-down work?

A. Yes.

Q. Going back to Exhibit 18, which is the lecture on microscopy data done in KCAC November 30, 1991, look at page TEM-4, which is under the heading of "Discussion," towards the bottom of the page, the second to the last paragraph.

A. Yes.

Q. All right.

Q. There they're talking about fibre width.

A. Yes.

Q. With respect to what they term fibre width, is that fibre diameter they're talking about there?

A. I believe so.

Q. And they talk about what the means and what the medians are?

A. Yes.

Q. And then they say, I'm reading at the

end, the last line, "all samples had their largest component in the range from 0.03 to 0.05 microns," as reported in table 4. Do you see that?

A. Yes.

Q. I guess that means just what it says, they --

Q. Most of the fibre diameters -- if I can summarize, most of the fibre diameters they found of the Calidria asbestos with the TEM analysis was in the .03 to .05 microns?

A. Yes.

Q. The reason I was asking that, when I was looking back at Dr. Addison's criticism of the Yeager paper, which is Exhibit 15, on page 2, one of the things he criticizes is the fact that Yeager makes his calculation assuming an average fibril diameter of 0.0333 microns, do you see that?

A. Yes.

Q. I understand that to mean that Dr. Addison thought that was not a proper calculation to use, is that what he appears to be saying?

A. I don't think so.

Q. Would you agree with me in the Yeager

paper the average fibril diameter was reported or was -- or assumed to be .0333 microns?

A. I don't have the paper in front of my. According to what Dr. Addison said, yes, that's the case.

Q. If you read further in that paper, Dr. Addison appears to be critical of the assumption that the average fibre diameter is .0333 microns, he says because the assumption made --

A. Where are you reading did.

Q. The last line, "would have an influence on the calculated fibre numbers per milligram."

A. That's what he says.

Q. Now, let me refer you to Exhibit 18 on table 2, which is at page TEM-7. And there -- they give a mean and a median for these Calidria asbestos fibers, all right, for these samples.

A. Yes.

Q. With respect to the median length, are those values generally consistent with other published size distribution data that you have seen?

A. I have to check again.

Q. I want to ask the same question with respect to table 3 at page TEM-8, which is fibre length breakdown or distribution. Are those generally consistent with other published data you've seen?

A. I've only seen RG-144. It's basically consistent.

Q. Are you generally familiar with the different grades of the Calidria asbestos and how they're processed?

A. To some extent, yes.

Q. Does this table, table 3, generally seem to indicate that the more the Calidria asbestos is processed or the greater processing it undergoes, the greater the number of fibers, number one, and the great number of fibers less than five microns, number two?

A. Say that again, the greater the processing, the higher the percentage --

Q. The higher percentage of short fibers?

A. It suggests the opposite, doesn't it.

Q. Let me ask it this way. Can you draw

any conclusions as to what table 3 suggests in terms of the more milling, what happens?

MR. WILL: When you say milling, processed in the mill?

MR. BROWNSON: Right.

A. I don't recall the precise details as to how one transforms RG-144 to RG-244, but I think there's a siliconization there. I don't think it refers to any more milling.

Q. Do you hold any opinions as a general matter how milling affects Calidria chrysotile in terms of size distribution?

MR. WILL: Can you be more specific when you mean by milling? As you know, there have been some experiments that involve ball milling, which is what goes on in the mill in King City in terms of processing this ore?

MR. BROWNSON: Process milling?

MR. WILL: In the plant in King City.

A. I don't know anything about it.

Q. That's all I have on this exhibit.

I want you to look next at Exhibit

number 19, which is the second fibre size determination and distribution paper done for KCAC, this was done in 1993, and it involves testing of RG-244, one of which was treated with silicon and one was untreated. Have you had a chance to review this paper?

A. I've haven't looked at it in great detail.

Q. Do these two papers, Exhibit 18 and 19, form a basis of any of your opinions in this case?

A. They may. I need to go over them in more detail.

Q. But at least at the present time, they do not?

A. I don't think I can say anything at the present time.

Q. Would it be fair to say if they do form a basis of your opinion, you don't know what that is?

A. I think -- my impression particularly with respect to Exhibit 18 is that the sample that I've become familiar with most, the RG-144, doesn't indeed have a rather small percentage of

fibers that reflect microns. That seems to be confirmed by Exhibit 18, and that the widths generally going between 3 and 500 angstroms is not that different from what I envisage as the size range for Coalinga fibre widths. I think I would like to be able to say yes. I would rely on this to say it's consistent with other data that I have seen.

Q. So in terms of these two exhibits, 18 and 19, there's nothing in here that startles or surprises you in terms of their conclusions for these size measurements and size distributions?

A. I would have to study it a bit more. There is the RG-244, which is in the table 3, Exhibit 18, it shows 11 to 18 percent fibers greater than five microns and 57 percent fibers greater than 10 microns. I found that a bit high, but I don't know much about the RG-244.

Q. And you say you find that a bit high because it's higher than what you seen in RG-144 samples?

A. Yes.

Q. Now on Exhibit 19 on the front, there's some stuff blacked out there. Is that

something you did or is that the way it came to you?

A. That's something -- I had some funny scribbling there.

Q. And did the funny scribbling have anything to do with editorial comments on your part?

A. No. It's just some odd marking.

Q. All right.

A. I remember something. Could I say something with respect to the threshold characterizations I made with regard to Coalinga asbestos? Just to simply indicate that it's not only my belief that one would have to work for, say, 600-odd years under that situation, it's also my belief that it may in fact be much longer than that because the calculations which I mentioned were based on, say, standard Canadian type of fibers. The Rochedale, I assume that's -- that's either a Canadian or a long South African fibre. I think what's worth commenting upon is that fact this the Calidria fibre or fibril in my opinion is equally different from the Canadian in being much more likely to dissolve, being much more soluble

than the so-called standard Canadian fibre.

Q. Is that a function of its chemistry or its size?

A. It could be both. It's certainly in my opinion the function of its size in that the vast majority of Coalinga or Calidria fibers are actually single fibrils. In that sense they're a lot thinner and in theory would be a lot more susceptible in the dissolution in the body fluids. There may be peculiar chemical differences and physical -- and chemicals are in a sense interlink features. But there's certainly evidence in data in my opinion to suggest that the Canadian fibers composite of at least six or seven fibrils cemented together with a matrix of unknown character, it's often regarded as a silicate of some sort. But the Calidria so-called fibre for all purposes is a naked fibril about no amorphous cementacious material.

Q. So if I can summarize what you just said, do I understand that you believe that the Calidria asbestos because it's shorter and thinner than UICC standard grade Canadian asbestos, it's less or requires longer to cause asbestosis

because it's more easily dissolved in the body?

A. If it were at all, plus these other properties of -- other properties lacking the inner fibril or matrix and other different properties.

Q. But one of those properties, as I understand it, is simply its size?

A. Yes.

Q. It's shorter and thinner, it's smaller in essence?

A. Yes.

Q. All right.

A. But I'm actually saying given equal lengths, say, of 7 microns in lenght, Coalinga fibre as opposed to a 7-micron lenght in Coalinga -- Canadian as opposed to Coalinga, it's my firm belief that the Coalinga fibre is much more susceptible to dissolution.

Q. If you look at Exhibit 18, let's take RG-144, which you say it is the type of Calidria you're most familiar with. It's indicated on page 7, table two, that the mean length of these fibers is 1.1 micron?

A. That's what it says are.

Q. For RG-244 ranges from two 2.45 to 3.22 in mean lenght?

A. Yes.

Q. If I understand what you just told us, that's shorter than standard UACC Canadian chrysotile?

A. I believe so.

MR. WILL: If you took a mean -- a mean is just an average?

MR. BROWNSON: Right.

Q. There's a media reported here, but I was talking about the mean which is average. Median means middle and mean means average in layman's terms?

A. Yes.

Q. Are you familiar --

A. The percentage -- just to clarify, the percentage of fibers in a UICC sample to my recollection, would be on the order of 25 percent and above where the percentage of fibers of fibrils and Coalinga above 5 microns would be in the order of 1 to 4 percent or 1 to 6 percent, according to the literature.

Q. You're talking about the size

A. I'm not familiar with -- I don't believe I have read it. Could I see a copy of it?

Q. Yes.

A. All right.

Q. What they're doing, they're discussing Dr. Dement's data from the textile plant in Charleston, South Carolina.

The first question I wanted to ask you, I'll wait -- have you ever read their paper before?

A. I don't believe so.

Q. If you look at the second page, which is --

Q. First of all, are you familiar with Rood and Streeter of the Occupational Medicine and Hygiene Laboratories, health and Safety Executive, London, England?

A. I've heard of Dr. Rood at the HSE, I don't know them personally.

Q. Are these the same Health and Safety Executive that you were talking about with the British amosite ceiling tile factory?

A. Yes.

Q. If you look at the second page of the

paper, which is actually page number 334.

A. All right.

Q. They say "Dement and Harris 1979 report several TEM-derived fibre size distributions for asbestos.

A. All right.

Q. We calculated the following from their data: in the chrysotile-using textile plant, for the processes of fibre preparation, twisting and weaving, the geometric mean lengths were 2.2, 1.8 and 2.0 on microns respectively." Do you see that?

A. Yes.

Q. First of all, have you ever seen that data reported before anywhere else?

A. I think so, but I don't recall.

Q. All right.

A. You mean the Charleston data?

Q. Right.

A. I think so, I think it's in Dement's thesis.

Q. If you look at page 336, which is table 1.

A. All right.

Q. It summarizes this data, and they show the mean and medium lengths of the fibers in these different, carding, weaving and spinning, operations. Do you see that?

A. Yes.

Q. Now first of all, you're familiar with the fact that the asbestos we're talking about is Canadian chrysotile asbestos?

A. Yes.

Q. And have you ever seen any different size distribution data other than what we're looking here on table 1 -- any different size measure data other than what we see in table 1 for the asbestos in the Charleston textile plant?

A. I believe so.

Q. And where is that data reported?

A. I don't recall.

Q. All right.

A. More in in terms of the percentage over 5, 10 and 20 microns.

Q. Right.

(Whereupon, document entitled "Characterization of Three Types of Chrysotile Asbestos after Aerosolization"

marked Plaintiff's Exhibit 21 for identification, as of this date.)

Q. We're now looking at Exhibit 21, which I think is a paper you're familiar with.

A. I'm familiar with it.

Q. So just to add some familiar ground. Exhibit 21 is a paper by Pinkerton and other authors characterizing three types of chrysotile asbestos after aerosolization?

A. Yes.

Q. You're familiar with this, of course?

A. Yes.

Q. One of the three types is what they call Coalinga mine chrysotile, but that's Union Carbide chrysotile?

A. I believe so, yes.

Q. And in the abstract and later in the body of the paper, they state, "The characterization of these chrysotile preparations in the aerosolized state, in particular the Coalinga Mine chrysotile, demonstrated different fibre length and fibre width distributions when compared with previous characterizations of samples that had been dispersed in a liquid medium

by ultrasonification"?

A. Yes.

Q. Do you see that?

A. Yes.

Q. I'm reading in the abstract.

A. Yes.

Q. Then it goes on to say, "These observations emphasize the importance of determining the size distribution of fibers in the aerosolized state for inhalation studies and the size distribution of fibers in a liquid suspension for oral ingestion, instillaton, or injection studies."

A. Yes.

Q. Finally say they, "Because of differences in length-width distributions, each of the studied chrysotile preparations would be expected to have different patterns of deposition in the alveolar regions of the lung after an inhalation exposure."

A. Yes.

Q. I want to ask you some questions about what we just read.

A. All right.

Q. In all of the fibre size distribution data that you have seen for Calidria asbestos, have you seen any size distribution data for that asbestos in an aerosol state, dust in the air other than what's reported in this paper?

A. I think the only other data set is Muhle, et al., 1987, I think it's Muhle and Pinkerton who have ever done that.

Q. Do you agree with the statement we just read that for purposes of inhalation studies, the size distribution in the aerosolized state is important?

A. Yes.

Q. And if I can put that in simple layman's terms, is that because inhalation studies of breathing asbestos, and obviously when you breathe something, what's important is what's in the air that you're breathing?

A. Yes.

Q. Now have you ever seen any animal inhalation data on Calidria asbestos?

A. Yes.

Q. What is that?

A. Pinkerton et al., Muhle, et al.

Q. And is that reported in your book?

A. Muhle, et al. certainly is.

Q. All right.

A. Pinkerton et al. may not be because I got it just a couple of years ago.

Q. I got a copy of your book here and I'm trying -- I want to go -- to the inhalation -- the compilation -- or the reference to inhalation data from the Calidria fibre. I don't know if we -- if you can find it higher.

A. Let me see.

A. It's on page 136.

Q. Let me put my tab there.

A. All right.

Q. And what you pointed out to us is the reference to the Muhle, et al. paper of 1987?

A. Yes.

Q. That's reported in your book as a chrysotile Calidria inhalation experiment on Wistar rats?

A. Yes.

Q. Have you read that paper?

A. Yes.

Q. And does that paper form any basis

for your opinions in this case?

A. Yes.

Q. An if you can tell us what is the fact or facts which you derive from that paper that form the basis for your opinion in this case?

A. To me it indicates Calidria chrysotile in high doses cannot induce tumors in rats.

Q. Is that because in 50 female rats, there were no mesothelioma tumors reported?

A. No tumors.

Q. All right.

A. No pathological changes at all.

Q. Of any sort?

A. Of any sort.

Q. Now with respect to Pinkerton's paper about inhalation, is that reported in your book?

A. No, it hasn't been reported yet.

Q. Is that because you're saying it was too recent to go into the book?

A. Yes.

Q. Now you also report at least three animal studies with respect to injection of Calidria asbestos in your book, is that correct?

A. Yes.

Q. And we talked about those a little bit last time, but I would like to ask you some questions about those and I'm not sure the best way to do it since I got the book. Maybe we have to look at it together.

A. All right.

Q. The first reference I see is page X.

A. Roman 10.

Q. Roman 10, I'm sorry. Table 3. Chrysotile 25 milligram, intraperitoneal injection of rats.

A. Yes.

Q. You report here Maltoni, et al. 1982.

A. Right.

Q. And it's listed as Calidria. My question is, is that Calidria?

A. I don't know.

Q. Have you read those papers?

A. Yes.

Q. As you sit here today, you don't recall whether it's Calidria or some other --

A. As I believe I said in the first deposition, I wrote to Maltoni and I said is it

California Calidria or Coalinga and he didn't reply.

Q. Is it your best understanding it's one of the Coalingas or it's fibre from the Coalinga deposit?

A. No, it could be -- there are a lot of different chrysotile deposits around Calidria.

Q. Now the other one I wanted to refer you was at page 90, that is the Suzuki paper 1984. That's actually is Calidria, at least as reported by you?

A. By Suzuki, it's RG-144 that he got from Art Langer.

Q. All right. Then we got at page 94, we got entries for Muhle, et al. which we talked about before, but now we're talking about injection and the inhalation. That is Calidria?

A. Right.

Q. And then we got Pott, and this is injection and that's Calidria?

A. Yes.

Q. And then we got Rittinghausen, et al., 1990?

A. All right. This is basically the

same lab.

Q. And that's Calidria?

A. Yes.

Q. Then, now on page 101, we got Maltoni again, but it's just recorded as Calidria chrysotile, we can't be sure what that is?

A. Right, that is the same study as I did in the beginning.

Q. Now I didn't find -- other than those injection studies we looked at and the inhalation study that you pointed to a minute ago, which I've now underlined by Muhle, is there any other animal data reported in your book which either are or could be Calidria asbestos?

A. There's one I found the other day with Maltoni and Minardi 1988 which I haven't actually seen before.

Q. Now that's reported in your index, but I didn't find it in your book here.

A. But again is Calidria. I don't -- on page 103.

(Discussion off the record)

Q. You've now identified on page 103 of your book that Maltoni and Minardi, which is

listed as Calidria chrysotile 1988 -- incidentally, it's listed as '89. Do you know if it's '88 or '89?

A. I believe it's '89. It came out in the IACC, but the galley I got was '88. You know how it is.

Q. Here --

Q. I have it here. We'll mark it here.

(Whereupon, document entitled "Non-Occupational Exposure to Mineral Fibres" marked Plaintiff's Exhibit 22 for identification, as of this date.)

Q. So now we've got Exhibit 22 and this is a photocopy I made of the 1989 Maltoni, et al.

A. Maltoni and Minardi.

Q. Yes. Injection.

A. Yes.

Q. That's reported in your book on page 102?

A. Right.

Q. What number is this?

A. 22.

a. Now what I was trying to figure out on this, and first of all, have you read their

paper?

A. Yes.

Q. It doesn't say anywhere in here that this is actually Calidria asbestos. And you had told us earlier that you asked Maltoni whether -- what this Calidria was with respect to the 1982. Did you ask the same with respect to this one?

A. I can't recall. I just called him in Bologna and his secretary answered and I said, "What's your fax number?" So I faxed him. I don't recall exactly what I put in the fax, whether I just said Maltoni '82 or in your studies using Calidria without actually specifying a paper.

Q. So you don't know as you sit here today, you haven't received a definitive answer whether this is Calidria or not?

A. No.

Q. If you look on page 49, under the heading "Materials and Methods." It makes a reference that the asbestos materials were obtained from Mount Sinai School of Medicine in New York?

A. Yes.

A. Yes.
Q. So to try to move this along, the actual laboratory research you have done concerning asbestos is shown in your CV in two places, one on page 2, second to last entry at the bottom, and then it appears again on page 7 in this entry in the middle from '85 to '87?
A. Are you asking me does this broadly apply to every -- let me say it differently. In terms of sitting in the lab and injecting rats, doing animal studies, specifically hands-on asbestos work?
Q. Right.
A. That's correct.
Q. I just want to ask some basic questions. The study of pathology is basically looking at tissue from the body, correct? That's what a pathologist does?
A. It's a compound question, really. What did you ask me, what was the first part?
Q. A pathologist, what a pathologist does is look at or study tissue samples, would that be fair to say?

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A. It could be tissue samples. It could be various fluids. It's a whole range of materials.
Q. Materials from the body?
A. A pathologist can also be an experimental pathologist. A pathologist can also be a research -- I think there is a whole array of categories of pathologists. The person who studies disease may be a fundamental research pathologist who never looks at a body, doesn't look at a whole organism, may spend his entire life doing in vitro studies or theoretical modeling.
Q. A pathologist does not treat living, breathing patients and put a stethoscope to their chest?
A. That's correct.
Q. The pathologist studies or analyzes samples of tissue and fluid and that sort of thing?
A. Pathology is the study of disease. A pathologist is a person who studies diseases.
Q. I'm not trying to make more of this than there is, but an epidemiologist also studies

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diseases, but a pathologist studies diseases from the perspective of looking at or studying or analyzing tissues and parts from the human body?

Q. First of all, have you seen that before?

A. Yes.

Q. And you had a chance to read it?

A. Yes.

Q. When did you first obtain a copy of this -- when did you first see this report?

A. I don't recall, nine, ten months ago.

Q. Is it fair to say that this is not included in your book?

A. Yes, it is.

Q. Is this report included in your book?

A. No.

Q. Why is that?

A. Too recent.

Q. So at the time you reviewed the literature and for this compendium which resulted in your book, you did not have a copy of this at your disposal?

A. No.

Q. Now let me ask you this, had you had a copy of this at your disposal, would you have included it in your book?

A. Yes.

Q. And where did you first get a copy of this exhibit?

A. Mr. Gerson.

Q. What is the time period during which you researched and compiled the literature that's tabulated in your book? When did you start and when did you finish?

A. Six, seven years.

Q. All right.

A. Probably more like five years, thinking about it.

Q. You mentioned that you obtained this Mellon Institute report, Exhibit 23 -- I'm wondering what was the cutoff date for material that went into your book?

A. '91.

MR. BROWNSON: Let's mark one more.

(Whereupon, Mellon Institute report dated September 3, 1971 marked Plaintiff's Exhibit 24 for identification, as of this date.)

Q. This is the September 3, 1971 Mellon Institute report.

MR. WILL: What date?

MR. BROWNSON: September 3, 1971.

Q. And I would like to ask you the same series of questions. This is not included in your book, is it?

A. No.

Q. And why not?

A. Same reason.

Q. Because you obtained it after you compiled the material for your book?

A. Yes.

Q. And did you obtain this report, Exhibit 24, at the same time you got the other Mellon Institute report?

A. Yes.

Q. And you got them both from Alan Gerson?

A. Yes.

Q. Now, you told us earlier that with respect to the six or seven with Mr. Bergstrom, Conwed cases dealing with mesothelioma, you did not believe that those mesotheliomas were caused by the Union Carbide asbestos because you just don't believe that could cause mesothelioma?

A. Correct.

Q. Is it your opinion that there is a background or threshold level of chrysotile asbestos exposure below which you simply cannot get mesothelioma?

A. I just said chrysotile cannot make mesothelioma.

Q. I'm just talking about chrysotile generally?

A. You mean like a tremolite contaminated.

Q. Let's talk about Canadian.

A. I believe there's a tremolite threshold from 200 to 300 fibre years.

Q. Is it your opinion that Canadian chrysotile by itself cannot cause mesothelioma under any circumstances?

A. In the absence of tremolite contamination?

Q. Yes.

A. I would say in light of my knowledge of the literature and study, yes.

Q. And is it your view that Canadian chrysotile asbestos as it exists with its tremolite contamination can cause mesothelioma but

there is a threshold below which it cannot?

A. Yes.

Q. In humans?

A. Yes.

Q. And do you base that in part upon the animal data which is reported in your book?

A. No.

Q. So the animal data reported in your book is not a basis for that conclusion?

A. No, it's more an understanding of the Thetford Cohort.

Q. So what you're talking about basically is studies by correspondence Corbet MacDonald up in Canada or Quebec?

A. Yes.

Q. Based on that data, you deduced that there is a threshold exposure to that Canadian chrysotile below which you cannot get mesothelioma and above which you can?

A. Yes. And and also the Libbyl vermiculytes and tremolyte data.

Q. It's also your view, is it not, that there's a threshold level of exposure to amosite asbestos below which humans cannot get

mesothelioma and above which humans can get mesothelioma?

A. Yes.

Q. Is that threshold based upon the animal data in your book?

A. No.

Q. It's not. What is that based upon?

A. Uxbridge Patterson Cohorts.

Q. That's based upon studies of workers --

A. Perhaps you could ask your question again to make sure.

Q. What I'm trying to find out, let's start with your opinion that there is a threshold or cutoff level of exposure to amosite asbestos below which humans don't get mesothelioma and above which they do.

A. Certainly the animal data contribute and support the idea that there is a threshold in humans, because in animals there are apparently levels beneath which one doesn't get mesothelioma. I was thinking more you were talking about the sort of specific figures we had derived, Kevin Brown and I, for amosite, the number of fibre

A. I don't know what the WDC samples had in terms of potential tremolite. I don't feel by any means that enough work has been done to exclude the possibility that there is an analogous situation in the animal studies, i.e. there's amphibole present. I think there has been lung burden analyses done of rats from Chris Wagner's study published in 1986, I don't remember, which have shown amphilbole in the lungs of these animals.

Q. How about the injection studies you reported in your book with Calidria asbestos where tumors were produced, are you saying that those tumors were not caused by the Calidria injected into those rats?

A. You were talking a second ago about Canadian --

Q. Now I've changed topics.

A. In terms of changed topics, I feel that tumors produced by Calidria in the Suzuki and Maltoni studies were due to exceedingly high dose, non specific mass effects predominantly.

MR. WILL: Is that assuming that Maltoni used Calidria?

THE WITNESS: Yes.

Q. With respect to Suzuki, you'll agree that the Suzuki injection studies produced tumors of mesothelioma. You're just claiming that they used too much?

A. Yes.

Q. A very high dose in essence?

A. Yes, with 25 milligrams minimum into the peritoneal cavities of a mouse.

Q. Going back to the question of amosite asbestos and this idea of a threshold or cutoff level below which will not produce mesothelioma and above which it will. As I understand it, that concept in your mind is based partly on the animal data and partly on studies of plant workers at the unarco plant in New Jersey and the Uxbridge plant in Britain?

A. Yes.

Q. Let me change gears a little bit and ask you about background levels of mesothelioma.

A. Right.

Q. Which you talk about in your book.

A. Yes.

Q. And again referring to the text of

the book, at the front of the book you make the statement, and I'm paraphrasing, you find a certain background or I guess idiopathic level of mesothelioma in animals?

A. Yes.

Q. And you translate that to humans, is that right?

A. Christ Wagner and I did compare idiopathic background types, in a sense non nonasbestos-related mesotheliomas found in humans. There's a certain degree of comparability.

Q. Let me put it to you this way. In reviewing this literature that you tabulated in your book, you have come up with this idea there's a certain number of mesotheliomas that occurred in animals that are not related to asbestos?

A. Yes.

Q. You say this lends support to the idea there are a certain number of mesotheliomas in humans that are not caused by asbestos exposure?

A. Yes.

Q. Is it your opinion that any of the seven cases at Conwed of mesothelioma were caused

by background or nonasbestos causes?

A. I have to look at the case.

Q. As you sit here today, you don't recall --

A. They're very detailed cases.

Q. You have stated in your book that in a particular animal study, if one mesothelioma is seen -- does not indicate to you that it's related to asbestos, is that fair to say?

A. Yes.

Q. Now you compile all of these different animal studies and the rates, whether they are injection or inhalation studies and the various percentages or rates of mesothelioma found, and what I'm wondering as a general statement, is there a level in animals, in the animal studies that you regard as background, be it 10 percent, 20 percent, 30 percent, whatever?

A. In my 1989 note to ASI, I discussed exactly that point. One mesothelioma in an inhalation study -- I think -- again, I would like to have the paper in front of me. I think it was 25 percent in the intraperitoneal study and somewhere 10 to 12 percent in the intraperitoneal

injection study.

Q. If we start with intraperitoneal injection studies in animals --

A. Yes.

Q. -- it's your general view, I'm not going to hold you to exact percentages, that about 20 to 25 percent of mesotheliomas that would show up in those studies are background or nonasbestos-related, is that fair to say?

A. Yes. In the context of the experiment.

Q. And do you make any sort of extrapolation or transference to human experience based on that fact?

A. Yes.

Q. And what is that?

A. To my mind it suggests that the number of asbestos fibers that need to be injected into the peritoneal cavity to produce a level which exceeds this level of background figure is extremely high.

Q. I don't follow you. You don't inject asbestos into the peritoneal cavity, you're talking about the animals?

A. I think I got the question wrong.

Q. My question is, if we take as a proposition your conclusion that the intraperitoneal studies show about a 20 to 25 percent background rate of mesothelioma in animals --

A. Yes.

Q. -- I'm wondering if we can take that percentage and use that as a basis that a certain percentage of human mesotheliomas are not caused by asbestos?

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

A. The problem is that the manner in which you arrive at the 20 to 25 percent figure is that you take the number of tumors which just simply arise spontaneously in the animal --

Q. Let me stop you there. You get that from the control group?

A. Yes.

Q. All right --

A. You get that -- from comparing the different amounts injected?

A. You get that from the untreated

control group.

Q. All right.

A. And then if you take the treated control group, in other words, a sham-treated control where you just inject the needle into the cavity, you get a certain percentage of tumors from that manipulation. If you then do a vehicle-alone treated control, so if the stuff is injected in saline solution, you just inject saline solution, you get another percent. Then if you inject what they call a nonfibrous dust alone control, you get another percent.

So this 20, 25 percent figure is the composite of the untreated control, the sham-treated control, the vehicle-alone treated control, the fibrous control, which is to focus on what specifically the agent is. That's the variation of the figures.

When you go to humans, the only element of that composite that is really comparable, since we don't inject things into animals, asbestos, would be the so-called untreated control. That I think is running about 1 to 2 percent. I would have to look at the data.

Q. Now which data would you look at to get that percentage?

A. I would look in humans, what data --

Q. In humans -- I thought that's what you said?

A. No.

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Q. Let's back up.

A. In animals, I would look at the so-called spontaneous peritoneal examples.

Q. Is that the 1 to 2 percent?

A. I don't recall what the number is. Or I would look at the untreated control group and I would say, well, there's a low level of mesothelioma arising in the peritoneal cavity of animals and there is indeed a certain percentage of peritoneal mesotheliomas arising in clearly nonasbestos-exposed humans. And it's very small.

Q. You say peritoneal --

A. We're talking about the peritoneal --

Q. We should leave the discussion there.

EXAMINATION BY

MR. WILL:

Q. I would like to ask you two follow-up clarification questions on this. Exhibit 18 and

Exhibit 20.

On Exhibit 18, Mr. Brownson asked you some questions about something on page 4 where it said that the -- all samples had their largest components in the range of .03 to .05 microns referenced in table 4. Does that indicate that the majority of the sample was in that range or can you tell from that statement alone?

A. I can tell.

Q. It could be just a plurality?

A. Yes.

Q. With respect to Exhibit 20, the Rood and Streeter article.

A. All right.

Q. Mr. Brownson asked you a question about the geometric mean out of a text. I'm referencing table 1. Does that also give the arithmetic mean?

A. Yes.

Q. And to your understanding, the tables in Exhibit 18 from ASTECO, are they geometric or arithmetic means?

A. I don't know.

Q. If you wanted to compare them, would

you need to know whether they are arithmetic or geometric, which to compare them to?

A. Yes. The most appropriate analysis would be a total fibre dimensional analysis.

Q. I'm just asking if you wanted to --

A. Apples to oranges.

Q. You have to compare arithmetic to arithmetic or geometric to geometric?

MR. BROWNSON: Unfortunately we didn't get that done, so we'll not conclude the deposition, but could we just ask that the reporter take the exhibits marked today and put them with the transcript, and I want a copy of the transcript. . We have five exhibits which are the five mesothelioma files -- we'll do it at the next session. We'll do it next time.

(Time noted: 4:30 p.m.)

_____

Subscribed and sworn to before me this_____day of _____, 1994.

_____

CERTIFICATE

STATE OF NEW YORK)
) ss.:
COUNTY OF NEW YORK)

I, DAVID OCANAS, a Certified Shorthand Reporter and Notary Public within and for the State of New York, do hereby certify:

That I reported the proceedings in the within entitled matter, and that the within transcript is a true record of such proceedings.

I further certify that I am not related, by blood or marriage, to any of the parties in this matter and that I am in no way interested in the outcome of this matter.

IN WITNESS WHEREOF, I have hereunto set my hand this 23 day of November, 1994.

DAVID OCANAS, C.S.R.

November 18, 1994

I N D E X

E X H I B I T S

November 18, 1994

E X H I B I T S (Continued)

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November 18, 1994

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IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF PENNSYLVANIA
----------x
In Re:
Asbesos Products Liability Litigtion (No. VI) Civil Action No. MDL 875
----------x
UNITED STATES DISTRICT COURT
FIFTH DIVISION
DISTRICT OF MINNESOTA
----------x
CONWED CORPORATION,
Plaintiff,
-against-
UNION CARBIDE CHEMICALS AND PLASTICS COMPANY, INC., (f/k/a UNION CARBIDE CORPORATION),
Defendant,
-and- Case No. Civ. 5-92-88
UNION CARBIDE CHEMICALS AND PLASTICS COMPANY, INC., (f/k/a UNION CARBIDE CORPORATION),
Third-Party Plaintiffs,
-against-
OWENS-CORNING FIBERGLAS CORPORATION, WALKER JAMAR COMPANY, A.W. KUETTEL & SONS, INC., API, INC., and MacARTHUR COMPANY,
Third-Party Defendants.
----------x

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June 21, 1994
10:25 a.m.

Deposition of EDWARD B. ILGREN, taken by Plaintiff, pursuant to Notice, at the offices of Kelley Drye & Warren, 101 Park Avenue, before Douglas M. Burke, a Shorthand Reporter and Notary Public within and for the State of New York.

0003

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0004

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ALSO PRESENT:
VIRGINIA RUSZCZYK

EDWARD B. ILGREN,
having been duly sworn by the Notary Public,
was examined and testified as follows:
EXAMINATION BY
MR. BROWNSON:
Q. I'm Bob Brownson. We are here today to take your deposition in a case entitled Conwed Corporation versus Union Carbide versus two or three third-party defendants.
I better state for the record that none of the third-party defendants are present here today. They were told about the deposition, so we are going to go ahead without them.
With that being said, as I understand it, you have had your deposition taken before, is that correct?
A. Yes.
Q. Have you ever had it taken in any context other than in asbestos litigation?
A. No.
Q. So it would be fair to say that any depositions that have been taken of you as a witness have been in the context of asbestos litigation, correct?

A. Yes.
Q. And that includes both personal injury and property damage litigation, as I understand it, correct?
A. Yes.
Q. Let me briefly run through those depositions. Before I do, first of all, you understand the deposition procedure, I know, but for the record is it clear to you what the procedure is here today and how the questions are asked and answers given, you understand that?
A. Why don't you just review it.
Q. Let me say this. First of all I'll ask questions and you can give answers and there's a couple of things I would like to say.

First of all, it's difficult for the reporter to take us both talking at once, so try to wait until the question is completed and I will wait until the answer is completed.

Secondly speak up audibly and don't mumble or shake your head or shrug your shoulders.

If any of the questions are unclear, tell me that before we answer them so we can be

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assured we have a record of answers in response to questions that you understood.

A. Okay.

Q. Let's go back to these prior depositions that have been taken where you were a witness.

With respect to personal injury asbestos cases, can you run down for me the depositions that you have given in those cases?

A. I believe it's Doherty versus W.R. Grace. The exact date I can't remember. Somewhere around two years ago.

The second and third were together, Ciliento and Seuss versus OCF.

The fourth would be Hermannson versus W.R. Grace/Cooper Industries.

I believe there are only four personal injury cases.

Q. The case of Doherty versus W.R. Grace, was that in Tampa, Florida?

A. Yes.

Q. In that particular case were you working on behalf of W.R. Grace?

A. Yes.

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Q. Any others or was it just Grace?

A. Just Grace.

Q. As I understand it, your deposition was taken in that case, but do you know if the case ever went to trial following that time?

A. Yes.

Q. Did it go to trial?

A. Yes.

Q. Did you testify at trial?

A. Yes.

Q. That was in Tampa, was it?

A. Yes.

Q. Do you recall on that occasion when you testified at the trial of the Doherty case whether that testimony was completed in one day or did you have to stay over?

A. One day.

Q. The other two cases, the Ciliento and Seuss, just so I'm clear, was your deposition taken in connection with both cases in two sessions or did they take it once for one of the cases and then a second time for the other one, or how did that work?
A. Two cases, one session, one day.

Q. So there was a single session of that deposition and it applied to both of those cases?
A. Yes.
Q. Where was that case pending, do you recall?
A. Jacksonville.
Q. Is that Duval County Circuit Court?
A. I believe so.
Q. On that particular case were you working on behalf of OCF or someone else?
A. OCF.
Q. In connection with this particular case, Conwed versus Union Carbide, let me ask have you done any work on behalf of OCF in this case?
A. No.
Q. Until you came here today were you aware that they were a third-party defendant in this case?
A. Yes.
Q. When did you first become aware of that fact?
A. I don't recall.
Q. In any event, going back to the case

in Jacksonville where the deposition was taken, do you recall if that case went to trial following the taking of your deposition?
A. Yes.
Q. Did it go to trial?
A. Yes.
Q. Did you testify at the trial?
A. Yes.
Q. Do you recall if that trial testimony all took place on one day or did that go over a second day?
A. One day.
Q. Were there defendants in this case other than OCF other than at trial, as far as you know?
A. Not that I recall.
Q. So as far as your best recollection today, there were the two plaintiffs and then defendant OCF?

A. Yes.
Q. And those three parties went to trial?
A. Yes.
Q. Who was representing OCF in that trial, do you recall?

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A. Mr. Richard Hines.
Q. Who is he with, what firm, do you know?
A. I can't recall.
Q. In preparing for your deposition or trial testimony in that case, did you meet with any attorneys other than Mr. Hines?
A. I don't recall.
Q. Do you recall if Mr. Garrard was involved at all in the preparation of that trial?
A. No, he wasn't.
Q. How about Mr. Shaw from Columbia, South Carolina, was he involved, Bruce Shaw?
A. No.
Q. Jim Crosby, was he involved?
A. No.
Q. The third case you mentioned is this Hermannson case. That was a case in the Cook County Circuit Court in Chicago, is that correct?
A. I believe so.
Q. As I understand it, your deposition was taken in that case prior to trial, is that correct?
A. Yes.

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Q. You also testified during the court proceedings at trial in that case?
A. Yes.
Q. I was unclear, but was a second session of your deposition also taken just before you testified in that case or how did that work?
A. I think a month before.
Q. Maybe I can help you out. I didn't bring it with me today, but I got a transcript of your deposition that was taken in the case. It seemed to indicate that there had been a prior session but you couldn't tell by reading the comments, and that's where I got the idea that maybe there was another session.
Does that help you at all recall whether there were two sessions or one?
A. There were two sessions.
Q. So you recall one being about a month before trial?
A. Yes.
Q. And then the second one on the eve of

trial?
A. No, sir.
Q. When was the second one in relation to
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Ilgren
the trial?
A. I believe a month before.
Q. So the first, then, would have been sometime about a month before trial?
A. Yes.
Q. Do you recall why it was they had a second session of that deposition? They just didn't get done?
A. I believe I was initially retained by W.R. Grace and they settled and then I received a call from the representative of Cooper, and she said we would like you to continue to work with us on the case.
Q. I understand you prepared two written reports in that case, is that correct, do you recall that?
A. I recall one. I don't recall two.
Q. Let me see if I can move this along. I saw a copy of one report you issued in the case which was a report addressed to, I believe, the lawyer for Grace, and then following that time you sent some either tumor tissue or clear lung tissue to Dr. Gibbs for some tissue burden analysis, and then there was much arguing and
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Ilgren
discussion among the lawyers on the record over whether that evidence could be admissible and there seemed to be a reference to a subsequent report about that, but I couldn't tell if there was or not. Do you remember?
A. I can't recall if a subsequent report was ever filed.
Q. Going back to these other three personal injury plaintiffs that you mentioned, Doherty, Ciliento and Seuss, do you recall if you had tissue examined by either Dr. Gibbs or Dr. Pooley in either of those three cases or for either of those three plaintiffs?
A. Possibly all three, though I don't recall exactly.
Q. Do you recall if it would have been Dr. Gibbs or Dr. Pooley or both?
A. I think it's Gibbs' and Pooley's lab.
Q. So --
A. To some extent they work together.
Q. Pooley is in Wales, correct, in Cardiff?
A. They are both in Wales. They are both

25 in Cardiff.
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1 Ilgren
2 Q. Do they share the same laboratory in
3 Cardiff?
4 A. I don't know. I just don't know their
5 physical setup.
6 Q. But in your experience, if you send
7 some tissue for a fiber burden analysis to that
8 lab, it could be done either by Pooley or Gibbs
9 or do you designate one or the other, or how does
10 that work?
11 A. Alan Gibbs receives the tissue and I
12 believe works with Fred Pooley to do the count,
13 the identification of the fibers, but I might be
14 wrong. I don't know.
15 Q. As I understand it, Dr. Gibbs is a
16 pathologist, is that correct?
17 A. Yes.
18 Q. And Dr. Pooley is not a medical
19 doctor, he is a mineralogist?
20 A. Yes.
21 Q. It's your understanding at least in
22 those three cases that they work together in
23 analyzing tissue, but you're not sure in those
24 three cases if it goes to Pooley first and then
25 Gibbs or vice versa, would that be a fair
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1 Ilgren
2 summary?
3 A. It goes to Gibbs first, but beyond
4 that I don't know.
5 Q. I'm going to jump ahead a little bit,
6 but in this particular case, Conwed versus Union
7 Carbide, do you know if any tissue of any worker
8 from the Conwed plant in Minnesota has been
9 examined by either Dr. Gibbs or Dr. Pooley?
10 A. I don't know.
11 Q. At least as far as any examination of
12 tissue being done through you, do you know if
13 that's been done or not by either Dr. Gibbs or
14 Dr. Pooley?
15 A. Not through me.
16 Q. So put another way, you have sent no
17 tissue to either Dr. Gibbs or Dr. Pooley for
18 analysis in connection with the present lawsuit,
19 would that be fair to say?
20 A. Yes.
21 Q. Have you seen any tissue samples,
22 whether they are blocks or slides, of any of the
23 Conwed workers involved in this particular case?
24 A. No.
25 Q. Have you seen any of the pathology
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reports of any of the Conwed workers involved in this case?
A. Yes, sir.
Q. Do yon recall the disease of the workers?
MR. WILL: You mean as alleged?
Q. The diseases alleged in the workers you have seen the reports of.
A. Alleged mesothelioma.
Q. How many of those have yon seen of the Conwed workers?
A. Six.
Q. We'll get into that later, but let's move on to the other depositions that you have given.
Other than the personal injury cases, I understand that you have worked in at least two building lawsuits concerning asbestos, is that correct?
A. Yes.
Q. Was one of them the Adams-Arapahoe School District case in Colorado?
A. I don't believe so.
Q. Do you recall the names of the two

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cases you were involved with in terms of giving depositions?
A. Cincinnati Schools, national Class Action and City of Baltimore.
Q. In the Cincinnati School case was that for GAF or was that someone else?
A. Celotex.
Q. In the National School Class action, who did you give a deposition on behalf of in that case?
A. I don't recall. Either Celotex or GAF. One or the other.
Q. In the City of Baltimore case, do you recall who the deposition was for in that case?
A. The same as the National Class.
Q. So it would be either Celotex or GAF?
A. I just don't recall:
Q. Let me ask you this: Do you recall if in the City of Baltimore case the product at issue that you were concerned with was floor tile?
A. I just don't recall.
Q. Other than those three cases, have you given any depositions in any asbestos building

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litigation?

A. No, sir.
Q. Have you testified at any trials in any asbestos building litigation?
A. No, sir.
Q. Have you given any depositions or testified in any asbestos insurance-related litigation?
A. No, sir.
Q. So would the depositions you have now told us about in the four personal injury plaintiffs and the three building plaintiffs comprise the depositions you have given?
A. I believe so.
Q. Likewise with the trials, it looks like you have testified in the three personal injury trials?
A. Yes.
Q. Would that comprise all of the trials that you have testified in?
MR. GERSON: Relating to asbestos?
MR. BROWNSON: Right.
A. Yes, sir.
Q. Let me change topics a little bit and

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talk about the present lawsuit.
My first question concerning this case is when were you first retained in connection with this case?
A. Perhaps two years ago.
Q. Do you maintain any sort of written record or file in connection with that, that would contain any materials in connection with this case?
A. A small file.
Q. Where do you maintain that file?
A. My office in Bryn Mawr.
Q. What are the contents of that file?
A. Records of alleged mesothelioma cases. Literature. Hygiene records. Related papers.
Q. Under the category of related papers, does that include correspondence?
A. Some.
Q. Is there a correspondence with any other medical doctors that you maintain in the file?
A. Yes.
Q. Is the correspondence between yourself

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and other doctors?
A. Medical doctors or doctors?
Q. Is there correspondence between you

and other medical doctors maintained in the file?
A. Who is a medical doctor? I don't mean to be silly or stupid.
Q. An M.D.
A. It doesn't have to be a treating physician?
Q. No, any M.D.
A. Related specifically to this case?
Q. Right.
A. Kevin Browne is an M.D., and that's all I remember at the moment.
Q. You didn't bring the file with you today to the deposition?
A. No.
Q. Did you review the file in preparation for the deposition today?
MR. GERSON: Which file are you referring to?
MR. BROWNSON: The file we have just been talking about that he maintains at Bryn Mawr concerning this case.

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A. At some point.
MR. BROWNSON: Trevor, do you have any objection to us getting a copy of his file?
MR. WILL: I think so. Can we be a little more specific? I don't think he reviewed the correspondence with Kevin Browne in preparation for the deposition, if that's what you're asking.
MR. BROWNSON: I don't know what he reviewed. Let me ask more about that.
Q. Let me zero in a little bit. As I understand it, you have a specific file you maintained in Bryn Mawr in which you keep papers in connection with this case, Conwed versus Union Carbide, is that fair to say?
A. I don't know if it's exactly specific to this particular case.
Q. You do have certain medical records specific to this particular case, correct, regarding --
A. Very few. Just the six case summaries.
Q. The six case summaries.
A. Yes.

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Q. Let me start with those. When you say you have six case summaries, are those summaries of records or are they actual medical records?
A. It's a sort of mixture, as I recall. Some are summary pages from medical records and

then one or two are like path reports and a radiology report. It's certainly not a voluminous, full record for each case.
Q. Do you know by whom the summaries of the records were prepared?
A. No.
Q. But in any event, at your office at Bryn Mawr in Pennsylvania you do have records concerning six Conwed workers alleged to have mesothelioma, would that be fair to say?
A. Yes.
RQ MR. BROWNSON: I request you to give us a copy of the records that he has concerning the six mesothelioma cases.
MR. WILL: Yes, I think we can get a copy of those.
Q. Other than the records of the six cases involving mesothelioma, do you have records of any other Conwed workers currently in your

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possession at Bryn Mawr or in that file or anywhere else?
A. No, sir.
Q. Have you seen records involving any other Conwed workers other than those six cases?
A. No, sir.
Q. Do you have any other plans that you're aware of at the present time to review records of any workers other than those six cases?
MR. GERSON: Conwed workers?
MR. BROWNSON: Right.
A. Perhaps.
MR. WILL: Given Fred Bergstrom's allegations, if we ever get any information on Fred, we may ask Dr. Ilgren to take a look at that. You know the situation with him. You're very aware of that.
Then depending on what your Dr. Kagan and Dr. Harber say, we may ask him for things in response to that.
Q. At the present time would it be fair to say that you have been asked to examine records of mesothelioma cases from the Colgate

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plant, as opposed to asbestosis cases, for example?
MR. GERSON: Beyond the six he said he examined? I don't understand your question.
Q. First of all, as I understand it, you have been asked to examine six cases, correct?

A. No.
Q. You have been asked to look at records of six cases?
A. No.
Q. What have you been asked to do in connection with those six cases?
A. Nothing.
Q. Have you been asked to make a determination as to the diagnosis on those six cases?
A. No, sir.
Q. Would you be able to determine the adequacy of the diagnosis based on the records currently in your possession or would you want further information to do that on those six cases?
A. I would have to review the individual

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cases again, but probably further information.
Q. Have you arrived at any conclusions, sitting here today, as of June 21, 1994, as to the diagnosis of those six cases?
A. No, sir.
Q. Have you spoken to any of the treating physicians on those six cases?
A. No, sir.
Q. By treating physicians, I mean to include pathologists who may have done stains or looked at tissues, have you spoken to any of those people in connection with those six cases?
A. No.
Q. Have you been requested to do so in connection with those six cases?
A. No.
Q. I understand from Mr. Will's comments, and he can probably correct me, but it's my understanding that Union Carbide has asked you to or has advised you that you may be looking at further Conwed worker records in the future.
Would that be a fair statement?
A. No, sir.
Q. So they have told you nothing in that

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regard?
A. No, sir.
Q. Other than what Mr. Will said here today?
A. Yes, sir.
Q. Have you seen any x-ray films from any of the Conwed workers?
A. No.
Q. Have you seen any of the pulmonary

function tests of any of the Conwed workers?
A. Perhaps, but I don't recall.
Q. If you had seen pulmonary function testing would it be of some of the six you have described or of someone else?
A. Of the six.
Q. So would it be fair to say that other than the six cases you have described for us, you haven't seen any of the records of the other Conwed workers?
A. Correct.
Q. Or summaries of the records?
A. Correct.
Q. Or x-ray films?
A. Correct.

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Q. Have you spoken to any physicians who have seen any of the Conwed workers either as consultants or as treating physicians?
A. No, sir.
Q. Have you been asked to speak to any of those physicians?
A. No, sir.
Q. Going back to this file you maintain at Bryn Mawr, is there an actual file that contains material specifically related to this case other than the medical materials on the six cases you have told us about?
A. Just a file.
Q. Does the file have a title?
A. No.
Q. Does it have any writing on the jacket or the file folder?
A. Just the names of the six.
Q. Of the six workers?
A. Alleged mesothelioma workers.
Q. In addition to that, have you collected any documents or other information relating to the Conwed case?
MR. GERSON: Objection to form as it's

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overly broad, but if you can answer it, go ahead.
A. I mean I don't want to annoy you, perhaps you could just qualify it a bit.
Q. Let me put it this way. You were retained approximately two years ago to work on this case, correct?
A. Yes.
Q. Since that time, other than the records of the six patients that you have described for us, have you collected any other

records or documents for your use in working on this case?
A. I believe I have some Conwed hygiene data, and there is some answers to interrogatories which describe the manner and type of asbestos used at the plant between 1958 and 1970, I think 1970 or '73.
Q. In connection with your work in this case, have you reviewed any literature, published literature, upon which you rely for any opinions you are rendering in this case?
A. Yes, sir.
Q. Is that maintained in some file?

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Where is that kept?
A. The same.
Q. That's in the same file that we were just talking about?
A. I guess not to be semantical, but it's not physically within the same file. It's within other file cabinets. It's a typical office with different file cabinets.
Q. Have you, for example, reviewed in connection with this case any of the published literature dealing with chrysotile fiber from the Coalinga mine in California?
A. Yes.
Q. Is that maintained somewhere in your possession?
A. Yes.
RQ MR. BROWNSON: I request a copy of the published literature dealing with Coalinga fiber that he has reviewed in this case.
MR. WILL: Let's go through this and let's discuss exactly how we are going to handle this. Let's do that and figure out the best way to get you what you need that's appropriate. I think you're going to want

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to talk to him about what else he has, so let's deal with this off the record and then put it on the record.
MR. BROWNSON: We can do that, but since I am a methodical person, my notes are in outline form, I'll just note them as we go along. I'm not doing it because I'm making multiple requests, but because that's the form I have.
Q. Have you also reviewed in connection with this case any unpublished documents or reports or studies concerning the Coalinga fiber from California?

A. Yes.
Q. Or analyses of that fiber?
A. Yes.
Q. Do you maintain copies of those reports or analyses?
A. Some.
Q. Do you maintain copies of all of the reports or analyses, unpublished reports or analyses of the Coalinga fiber that you have reviewed in connection with this case?
A. Yes.

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RQ MR. BROWNSON: I ask to get a copy of those too.
MR. WILL: Same thing.
Q. Do you recall what form those reports or analyses take?
A. Paper.
Q. They are on paper as opposed to computer disk, correct, or some other format?
A. Yes.
Q. Do you recall if those were reports or analyses which were done specifically in connection with this case or are they reports or analyses that were done in some other context that you obtained for use in this case?
A. Another context.
Q. Who were the authors of those?
MR. GERSON: Of all the unpublished?
MR. BROWNSON: Right.
MR. GERSON: You're asking about the unpublished reports which he reviewed in connection with this case?
MR. BROWNSON: Right, concerning the Coalinga fiber.
A. Off the top of my head, I'll do my

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best.
Q. Give the ones you can recall.
A. Nauman. Dresher. Woolery. Park. Jordan. Hodgson. Addison. Bright. Thickstun. Addison. Mumpton. Graf. Haartz. Martin. IITRI. Browne.
Q. Is that Kevin Browne?
A. Yes. Langer. Morgan, Arthur Morgan.
Q. Those are what you recall?
A. At the moment.
Q. Have you seen any --
A. Weiss. Then the MDH Conwed report.
Q. That's the Minnesota Department of Health?
A. Yes, 1989.

Q. That's March 1, 1989?
A. I don't recall. March or September.
Q. It's a thick report?
A. Yes.
MR. WILL: You're giving the published ones now?
THE WITNESS: No, unpublished.
Q. I guess you included that because I'm not quite sure if that's published or

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unpublished.
A. Published as in standard peer-reviewed, scientific, off the shelf, yes. They would be not in that category.
Q. That's the context in which I was asking the question. When I said published, I meant peer-reviewed publications.
A. Yes.
Q. Any others?
A. Again off the top of my head, I don't recall.
Q. In addition to the reports or papers that you have just described to us, unpublished reports or papers that you have reviewed in connection with this case, have you reviewed any other unpublished reports or papers dealing with Coalinga asbestos that you did not consider in connection with this case?
MR. GERSON: Referring to papers specifically about the Coalinga?
MR. BROWNSON: Right, which deal with it in some fashion. Let me put the question another way.
Q. Were there some papers or reports or

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analyses dealing with the Coalinga asbestos fiber of which you are aware and you looked at and you decided this just has no relevance to the Conwed versus Union Carbide case or I'm not going to consider it in the context of that case?
A. Not to my knowledge. The question is slightly complicated, but not to my knowledge.
Q. Let me ask you a little bit about the reports you have described.
First of all, the Minnesota Department of Health report, I actually brought a photocopy of it here and I'm sure it's the one you're talking about, a report to the Minnesota legislature and a picture of the Conwed plant on the front?
A. I don't recognize the cover.
Q. Actually in its published, and I use

that term in quotation marks, version, it's a brown or tan or yellowish cover. Does that help you?
A. Yes.
Q. So I have just shown you a report which is a photocopy of a report entitled "Medical Screening for Asbestos-Related Lung

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Disease among Conwed Corporation Cloquet Workers And Their Spouses, Preliminary Report To The Legislature, March 1, 1989," by the Minnesota Department of Health.
I will show you the photocopy of the report. Is that the report that you mentioned?
MR. GERSON: Do you want to mark that as an exhibit?
MR. BROWNSON: No, I don't. I do have a clean copy we could mark.
Q. Is this a report that you reviewed?
A. It appears to be so.
Q. They always look a little different in photocopy form.
A. Yes, it appears to be.
Q. Do you recall if the version that you reviewed and is in your possession is an actual bound version that has the yellow or tan cover on it?
A. No, as I recall it's unbound.
Q. In addition to that report, have you seen or reviewed any other materials from the Minnesota Department of Health in connection with the Conwed workers?

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A. I think one set of hygiene data came from the MDH. I don't recall.
Q. When you say one set of hygiene data, do you recall what sort of material was in that? Was it air samples?
A. Yes.
Q. Is that something that you maintain in your possession?
A. Yes.
Q. Do you recall that being some data that was compiled by the Minnesota Department of Health?
A. I believe so, but I don't recall.
Q. Did you receive it directly from the Minnesota Department of Health, as you recall?
A. No.
Q. Do you recall the date or approximate date of the air samples that were taken in that data?

A. No.
Q. Do you know if those samples were taken on or before 1974?
A. I think so, but I don't recall the exact date.

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Q. Have you seen any of the air-monitoring data taken by Union Carbide industrial hygienists at the Conwed plant in approximately 1972?
A. I believe so, yes.
Q. Is that the data you're speaking of or were you speaking of something different than that?
A. Different than what?
Q. You said earlier you had seen some hygiene data that you thought might have come from the Minnesota Department of Health. I'm wondering if that data is the data that was taken by the Union Carbide industrial hygienists in Cloquet in October '72 or if it's some other data.
A. I don't recall. It might be the UCC set. It might also be another set. I just don't recall.
Q. Do you recall, sitting here today, seeing any air sampling or air-monitoring data out of the Conwed Cloquet, Minnesota plant other than taken by Union Carbide industrial hygienists?

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A. I just don't recall.
Q. Would it be fair to say that whatever data you have seen, you have a copy of it in your possession somewhere?
A. Yes, sir.
Q. So when you spoke earlier about seeing hygiene data and having a copy of hygiene data, to use your term, is that what you're speaking of, or were you talking about something in addition to that?

MR. WILL: I lost the reference to "that."

Q. You have told us that you do recall seeing Union Carbide air-monitoring data from the Conwed plant, correct?
A. Yes.
Q. In addition to that, you thought maybe there was something else that you got through the Minnesota Department of Health, but you couldn't be sure.
A. Yes.

Q. My question is when you spoke earlier about having in your possession copies of hygiene data from Conwed, are those the materials you

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were speaking of or were you including something else?

A. Those were the materials.

Q. You also told us about interrogatory answers that you have seen and reviewed and you maintain in your possession.

A. Yes.

Q. Who was answering these interrogatories? Is it Union Carbide or is it Conwed or is it both or somebody else?

A. I don't recall. I think it was Conwed, but I don't recall.

MR. WILL: I can help you. They are your answers to interrogatories, the question that asks about the purchase of asbestos fiber.

A. There is also the NAAC letter to I guess Conwed.

Q. You're speaking of a letter from the North American Asbestos in Chicago?

A. Yes.

Q. To Conwed?

A. Yes.

Q. You have seen that letter and you

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maintain a copy of that letter in your possession?

A. Yes.

Q. In addition to that, as I understand it, you have some interrogatory answers that describe the shipments or use of asbestos to the Cloquet plant?

A. Yes.

Q. Included with that did you get copies of invoices showing shipment of fiber to the plant?

A. No.

Q. Sitting here today, can you give us a sketch of what you recall the use of asbestos at the Conwed plant based upon those materials?

A. A sketch of it?

MR. WILL: What do you mean, by date, by fiber type, by amount? Are you asking him to describe the production process?

Q. I'm asking based on the materials you have described to us, I want your understanding of what type of asbestos was used and when and how much, as you recall it.

A. The document is eight or nine pages long. The first half seems to list the chrysotile. The second half seems to list the amosite. The initial date of input for chrysotile I think was 1966, though I can't be sure, and it comes in the form of what they describe as cationic pellets, a few odd shipments of loose form, and I think there is another form again that seemed all to be Union Carbide.

Then it's broken down, if I recall, several installments per year from 1966 to, I think it's 1973.

I don't remember the tonnage per year, but there seemed to be from several hundred to several thousand.

Then the amosite began, as I recall, in 1958. It was from NAAC/Cape, I assume the Cape distributor. It was grades G, GK and D, I think. Again the tonnage, as I recall, ranged from several hundred to several thousand tons, though I wouldn't say that was per annum. Again I would have to sit down with the numbers.

Q. Let me ask you this. Have you obtained information concerning the amount and type of asbestos used at the Cloquet plant from

any source other than the interrogatory answers and the letter from North American Asbestos that you have described?

A. Yes.

(Recess taken.)

BY MR. BROWNSON:

Q. The last question was have you seen other documents concerning the use, amount, type or date, the use of asbestos at Conwed other than those that you have described to us?

A. I think so, but I can't recall the titles.

Q. Whatever those would be, would copies of those also be maintained in your possession?

A. Yes.

Q. In connection with your work in this case, you mentioned to us earlier that you had either obtained or reviewed or relied to some extent on correspondence with others.

MR. WILL: I don't think he said that.

Q. I thought you did, so let me ask the question.

In connection with your work in this

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case, Conwed versus Union Carbide, have you collected or obtained or used correspondence from other medical doctors, as we discussed before?
MR. WILL: That's actually kind of a multiple question. I'm not trying to be too difficult.
A. You asked me about Kevin Browne.
Q. You said there was some correspondence between yourself and Kevin Browne.
A. Yes.
Q. Is that correspondence to Browne or from Browne or both?
A. I don't recall. I think it's both. It could be both.
Q. Is that correspondence that was generated in connection with your work on this case?
A. Perhaps not specifically.
Q. Was it correspondence that was generated in connection with the issue of Coalinga chrysotile fiber?
A. In part, I think.
Q. Does the correspondence mention Coalinga chrysotile fiber?

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A. I think so, but -- I said before I thought so. I'm not a thousand percent sure.
Q. Is it something that you relied upon in any way in your work in connection with this case, that correspondence?
A. No, sir.
Q. Is it something you relied upon in any way in connection with any opinions you hold as to the biological potential of Coalinga chrysotile asbestos?
A. I think it's safer for me to actually look at the correspondence, because I can't recall at the moment the nuts and bolts of the contents.
Q. Have you spoken to Dr. Kevin Browne concerning this case, Conwed versus Union Carbide?
A. Not specifically this case.
Q. Have you spoken to Dr. Browne concerning the issue of any biological potential or health effects of Coalinga chrysotile asbestos fiber?
A. Possibly.
Q. Do you know if you had any record of

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those conversations? Has that been reduced to

writing in any way?
A. I don't think so, but possibly.
Q. In addition to this correspondence you mentioned between yourself and Dr. Brown, do you have any other correspondence with anyone that deals with this particular case that you maintain in your possession?
A. Could you repeat the question.
(The record was read.)
A. There are a couple of letters from Trevor Will.
Q. To you?
A. I believe so.
Q. Other than those couple of letters from Trevor Will to you, do you know of any other correspondence that you have in your possession in connection with this case?
A. Nothing springs to mind.
Q. Do you know if there has been any correspondence generated in connection with this case that may no longer be in your possession?
MR. GERSON: Generated by anyone?
MR. BROWNSON: Right, that he has seen

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Ilgren

in connection with this case.
A. Off the top of my head, no.
Q. You had mentioned earlier that you maintain in your possession copies of what you described as related papers and you indicated that correspondence was one of the things that would be a related paper.
Other than correspondence can you think of what else these related papers would comprise?
A. Papers related to Calidria.
Q. Just for the record, Calidria is a trade name that Union Carbide used and maybe successor companies have used for the Coalinga asbestos that comes from the New Idria deposit, is that correct?
A. I just thought, it may be incorrect, I thought the derivation was Cal for California and Idria for the Idria serpentinite.
Q. So our record is clear, we have been speaking at this deposition about Coalinga. You mean Coalinga asbestos and we have been speaking of Calidria, and those are one and the same thing, is that correct?

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MR. GERSON: I don't recall that I recall your question referred specifically to Union Carbide Calidria. I think your

question was with respect to Calidria generally.
A. I would say that there is a Calidria fiber that may have been mined by Union Carbide or JM or Atlas or not mined, just left in the mountain. I use them interchangeably, Coalinga fiber and Calidria fiber, I use them interchangeably, not with reference specifically to Union Carbide.
Q. And that's what caused the confusion.
A. Right.
Q. You are aware that there is this serpentine deposit which is the New Idria deposit which is sometimes called the Coalinga deposit, and it's referred to as Coalinga fiber, is that correct?
A. Yes.
Q. In that deposit in California which Trevor Will has visited, as I understand it, there have been over time three operating mines or pits and one is the Union Carbide, one is the

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Ilgren

Johns-Manville and one is the Atlas, is that correct?
A. Probably one or two smaller, but that's correct.
Q. When you use the term Calidria, you use that term generically to mean the Calidria deposit, and it could be fiber from actually any of those three commercial mines?
A. Yes.
Q. In addition to that, and this is maybe what caused the confusion, Union Carbide uses Calidria with a capital C as a trade name or did for some of its asbestos.
A. I didn't know.
Q. Were you aware of that?
A. I don't believe so.
Q. Let me back up a little bit and ask you about the unpublished papers, reports or analyses that you have reviewed and you have in your possession that you listed for us, and I want to just run through them and have you briefly describe for me what they are.
Let's start, you mentioned Weiss. Is that a report by Dr. William Weiss?

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A. Yes.
Q. He is a medical doctor in Philadelphia?
A. Yes.
Q. Is that a written report?

A. Yes.
Q. Does it deal specifically with this case or does it deal with more general issues?
A. Conwed hygiene, Conwed MDH survey.
Q. Is that a commentary or a review or a critique or an analysis of the Minnesota Department of Health data that's been prepared by Dr. Weiss?
A. Yes.
Q. Do you know what the date of that is?
A. Precisely, no. Recent.
Q. Does it take the form of a letter or a report or --
A. To be specific, I haven't actually, I don't posess a copy. He and I discussed it. I have taken some notes per his various criticisms, but I don't actually physically have a copy of the critique.
Q. Do you maintain a copy of the notes

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Ilgren

you have taken per your discussions with Dr. Weiss?
A. Yes.
Q. In addition to those notes, have you seen any sort of writing prepared by Dr. Weiss in connection with his critique of the Minnesota Department of Health report?
A. No.
Q. So what you have had is oral conversations with Dr. Weiss?
A. Yes.
Q. Have the two of you actually sat down face to face and discussed his thoughts and critiques of the Minnesota Department of Health report?
A. Yes.
Q. Do you recall when that occurred?
A. Months ago.
Q. Was it done in connection with this particular lawsuit, in whole or in part?
A. In large part.
Q. In addition to this particular lawsuit, did that conversation concern any other cases, legal cases or claims on which you are

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Ilgren

working concerning the Calidria or Coalinga fiber?
A. No, sir.
Q. In addition to this lawsuit, have you ever done any consulting work or other type of work in connection with a case or a patient or a claim involving exposure to Calidria fiber?

A. I believe there was one, but I can't recall the name, a few years ago.
Q. Do you recall if it was a case of a worker from the Conwed plant?
A. No, it wasn't.
Q. Were you aware of the fact that many of the Conwed workers in cases separate and distinct from this one have brought legal cases for injuries as a result of exposure to Calidria fiber?
A. Would you read that back.
(The record was read.)
A. Not specifically, no.
Q. First of all, are you aware of the fact that in this particular lawsuit, Conwed is suing Union Carbide to try to collect reimbursement for past and future workers

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Ilgren

compensation costs? Are you generally familiar with that claim?
A. Yes.
Q. What I'm asking is are you aware of separate lawsuits brought by actual workers against Union Carbide separate from this lawsuit where they're trying to seek recovery for injuries they claim were as a result of exposure to Calidria asbestos?
A. No, sir.
MR. GERSON: Were you asking if he is aware of the particular cases or aware generally that such cases exist?
MR. BROWNSON: That's a good distinction.
Q. Were you aware generally that such lawsuits existed?
A. No, sir.
Q. Have you ever worked on any particular case brought by any worker alleging injury as a result of exposure to Calidria asbestos from Conwed?
A. No, sir.
Q. Have you ever worked on any cases

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Ilgren

brought by any worker alleging injury as a result of exposure to Calidria asbestos at any other place other than Conwed?
A. Yes, sir.
Q. Was that the one you just mentioned a minute ago?
A. Yes, sir.
Q. Do you recall if that was one case or more than one case?

A. I believe one case.
Q. Were you working on behalf of Union Carbide in that case or somebody else?
A. Union Carbide.
Q. Do you recall where that case was located?
A. I think it was Texas. I don't recall.
Q. Do you recall if that case arose out of a Georgia-Pacific wallboard plant in Texas? Does that ring a bell at all?
A. No bells.
Q. Do you recall what work it was that you did in connection with that case?
A. I don't recall specifically. Just reviewing a lot of medical records, perhaps

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seeing some path slides.
Q. Do you recall if that case was a mesothelioma?
A. No, sir, I don't recall the details.
Q. Do you recall if it was a cancer of the larynx?
A. I just don't recall.
Q. So basically sitting here today, you don't recall what disease was either involved or alleged to be involved in that case?
A. No, sir.
Q. Do you recall seeing any tissue in that case, either slides or actual samples of tissue?
A. No, sir.
Q. Do you recall seeing any reports from Dr. Pooley in that case?
A. No, sir.
Q. Do you recall if Dr. Pooley was involved in that case?
A. No, sir.
Q. Do you recall asking Dr. Pooley or Dr. Gibbs or anyone else to do any fiber burden analysis in that case?

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Ilgren

A. No, sir.
Q. Do you know if either Dr. Gibbs or Dr. Pooley did any fiber burden analysis in that case?
MR. GERSON: I object to relevance. That case is that case and we are here for this case, so I don't quite see the connection. You can answer, but I want to put that on the record.
A. No, sir.
Q. Other than yourself, are you aware of

any other consultants who may have been working for Union Carbide in that particular case?
MR. GERSON: Same objection.
A. No, sir.
Q. Do you recall discussing that case with any other consultants who may have been working for Union Carbide?
A. No, sir.
Q. Do you recall preparing any critiques of the work of anyone else in that case, experts for the plaintiff or other defendants?
A. No, sir.
Q. Do you know with whom you were working

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on behalf of Union Carbide in that case?
A. Yes, sir.
Q. Who was that?
A. Gary Elliston.
Q. Mr. Elliston, is he with Henry Garrard's office?
A. No.
Q. Where is he from?
A. I don't recall.
Q. Is he from Texas?
A. Yes.
Q. Other than that particular case and the present case, have you done any consultation work of any type for Union Carbide in connection with Coalinga asbestos?
A. Yes.
Q. Can you describe for us what that consulting has been or that work has been?
A. Yes. Just an ongoing analysis of literature relevant to the biological, physical and chemical characteristics of Calidria.
Q. When did that begin?
A. On behalf of UCC?
Q. Right, on behalf of UCC.

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Ilgren

A. I don't recall exactly. Four years ago, five years ago.
Q. So would it be fair to say that by the time you were first retained or did your first consulting in this case, you had already been doing some consulting with Union Carbide in connection with the Calidria asbestos?
A. Yes.
Q. By whom were you retained initially to do that consulting?
A. Mr. Gerson.
Q. I assume that from time to time you have had discussions with Mr. Gerson about what

you're doing, you report to him and that sort of thing?
A. Occasionally.
Q. Other than Mr. Gerson, is there anyone else to whom you have reported concerning your consulting work for Union Carbide with respect to the Calidria asbestos?
A. Not that I recall.
Q. Are you on some sort of retainer for that work or do you just do work as requested and then charge for it or how does that work?

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Ilgren
MR. GERSON: Objection. Go ahead and answer.
A. As requested.
Q. Going back over the four years or so that you have been doing this consulting work, can you describe for me the type of projects or work that you have done in connection with the Calidria asbestos?
A. It's not a hyper-defined, mandated project. Just an ongoing analysis of the biological, physical, and chemical characteristics of Calidria per existent literature.
Q. If I could summarize this in a short capsule, which I'll attempt to do, would it be fair to say that about four years ago you were asked by Mr. Gerson to monitor the literature concerning Calidria asbestos and stay on top of it and analyze that literature as it comes out?
A. Yes, sir.
Q. In connection with doing that, as I understand it, you have actually become involved in at least two specific lawsuits, one is this one and one is that one from Texas?

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A. Yes, sir.
Q. Other than those two, can you recall any other specific claims or lawsuits which you have been asked to work on in connection with the Calidria asbestos?
A. No, sir.
Q. Have you been asked to do any laboratory research in connection with the Calidria asbestos?
A. No, sir.
Q. Have you done any laboratory research in connection with the Calidria asbestos?
A. No.
Q. Have you requested anyone else to do any research in a laboratory with respect to the

Calidria asbestos?
A. Yes.
Q. Who is that?
A. Hartwig Muhle. Arthur Morgan. Walter Eastes. Don Rimstidt. That's all I can recall.
Q. Let me just go through those. What did you ask Hartwig Muhle to do?
A. To regenerate the Calidria or Coalinga dust cloud in a manner similar to the way he did

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it in 1987.
Q. Are you familiar with the paper that he was one of the co-authors on which talked about generating an aerosol dust cloud from, among other things, Calidria asbestos?
A. Yes.
Q. Are you saying that what you asked him to do was do that again?
A. Yes.
Q. Why did you make that request?
A. I wanted to understand why he had only 130 fml out of 6 mg/m3.
Q. Are you saying in his paper he got 130 per fml?
A. Circa, about that.
Q. In his aerosol dust cloud from a certain mass of Calidria fiber?
A. Yes.
Q. Basically you were asking him to replicate that, to see if those results would be duplicated or not?
A. Yes.
Q. Did you ask him to use any different technique or the same technique?

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Ilgren

A. Same.
Q. Was that done?
A. No.
Q. Do you have any knowledge or indication that it will be done?
A. Yes.
Q. Is the work underway?
A. Perhaps.
Q. Do you know if it is or not?
A. Probably, but I don't know.
Q. Do you expect that work is going to be done?
A. Yes.
Q. In other words you have gotten the go-ahead or given him the go-ahead to do the work and you expect that he is going to do it?
A. Yes.

Q. Do you expect that there will be any written report generated as a result of that work?
A. I don't know.
Q. Have you asked him to prepare any report as a result of that work?
A. No, sir.

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Q. Where is he doing this work?
A. Hanover, Germany.
Q. That's at his laboratory?
A. Yes. It's not specifically correct to say he is doing it. He may do it.
Q. Where would you expect the work to be done?
A. Hanover, Germany.
Q. Why are you interested, jumping ahead, why are you interested in the aerosol generated by Muhle?
A. It failed to produce any pathological lesions in the rats.
Q. You are referring to his inhalation studies?
A. Yes.
Q. From the aerosol generated with a number of materials, including Coalinga asbestos?
A. Yes.
Q. On that topic, you're again speaking of these inhalation studies he did as published in, I think it was published in '88, is that right?
A. I have the galley proof he gave me in

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'87, so it may have come out in '88. I wouldn't be surprised.
Q. I have it here, but I'm trying to move along here. He has published once in the peer review medical literature concerning these inhalation data, has he not?
A. Yes.
Q. So whenever that turns out to be, that's the one we are talking about, around '87 or '88?
A. I believe it's only once. I believe that's the case. Annals of Occupational Hygiene.
MR. BROWNSON: Muhle, Pott, et al., Annals of Occupational Hygiene by the British Occupational Hygiene Society.
Q. Is that the one we are talking about?
A. Yes.
Q. I highlighted here, it says as a

result of a conference in October '86, and the publication date looks like 1987?
A. Yes.
Q. You mentioned that you have asked Dr. Muhle to do some work and we have talked
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about you requesting that he regenerate this aerosol. Have you asked him to do any other work?
MR. GERSON: In relation to Calidria?
MR. BROWNSON: Right. All these questions deal with Coalinga.
A. Possibly finalize the lung burden analysis, same study.
Q. When you say possibly finalize, have you actually asked him to do something or you might have asked him and you don't remember?
A. I think I asked him and then unasked him and then I can't remember whether we decided to go ahead and ask him again.
Q. Do you know if that work is underway?
A. It's not underway.
Q. Do you have any expectation that that work would be done in the next year or so?
A. I just don't know.
Q. What was it that you were considering when you made that request or may have made that request? What was the question that you were asking?
A. Potential retention of Calidria
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chrysotile in the lung, is it retained.
Q. Were you asking that he go back and look at tissue from the rats in his 1986 inhalation study?
A. Yes.
Q. Basically you asked him to go look at that same tissue, as opposed to do another inhalation study and take a burden from the new rats?
A. Yes.
Q. Have you asked him to do anything else?
A. Not that I recall.
Q. You mentioned you have asked Arthur Morgan to do some work. What work have you asked him to do?
A. Differential solubility studies.
Q. Of what, Calidria fiber as opposed to something else?
A. Yes.
Q. What's the something else?

A. Other chrysotile types.
Q. Have you asked him to do a differential solubility with respect to specific

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Ilgren

other chrysotiles such as like a UICC/A or B or any chrysotile at his discretion or what?
A. He counter-proposed with a series of other types. I don't recall which ones. AA, B, F, UICC/B.
Q. This is solubility in what medium?
A. I think his system is 25 degrees centigrade, 1 normal HCL.
Q. Are you aware of any work that Arthur Morgan has done to date concerning the solubility of Calidria compared to other types of chrysotile?
A. No.
Q. So when you spoke earlier about papers or reports you have seen from Arthur Morgan, that was something else, this question of solubility of Calidria as opposed to other chrysotiles?
A. Yes.
Q. Do you know whether that work has been done yet by Dr. Morgan?
A. It has not.
Q. Do you know if it's underway?
A. It's not underway.
Q. Do you know if it will be underway in

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Ilgren

the next year or two?
A. It may be, yes.
Q. Have you asked Dr. Arthur Morgan to do any other work other than that differential solubility?
A. Not that I recall.
Q. How about Walter Eastes, who is Walter Eastes?
A. He is another in vitro Morgan-type. He works in Ohio.
Q. I'm not familiar with him. Where is he located in Ohio?
A. I can't remember. Grandville someplace.
Q. Is he affiliated with some university?
A. I would have to go check. He is a colleague of Arthur Morgan's. He has a physiological system rather than an HCL system for looking at differential solubility.
Q. Do you know has he done any work to date with respect to Calidria asbestos of which you're aware?
A. No, sir.

Q. As far as you know, your request to

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him that he do some work with respect to Calidria would be the first work he has done on that fiber?

A. Yes, sir.

Q. Do you know has that work been done?

A. No.

Q. Has it been started?

A. No.

Q. Do you expect it to be started in the next year or two?

A. Hopefully.

Q. Has Union Carbide given the go-ahead to do that project?

A. No, sir.

Q. Is this a project that you have requested that Union Carbide undertake or is this something you're doing independently on your own?

A. The former.

Q. So the request has been made of Union Carbide, you just haven't gotten the go-ahead yet?

A. Yes.

Q. Is the same true with the solubility, differential solubility study by Arthur Morgan?

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A. Yes.

Q. You have made the request to Union Carbide but have not yet gotten the go-ahead?

A. Yes.

Q. If you did get the go-ahead, how long do you anticipate it would be before you get results from either of those two studies?

A. I really don't know. Hopefully within a year, but I don't know.

Q. I suppose that's dependent on many things, such as how busy they are and that sort of thing. I'm just asking for some ballpark estimate.

A. Sure.

Q. You mentioned Don Rimstidt. Can you describe who he is?

A. I don't know how you would label Rimstidt, a something-ologist. I don't know whether he is specifically a mineralogist or geochemist, but he models the rate of chrysotile dissolution using certain computer software in his so-called shrinking fiber model.

Q. Do you know has he done such modeling to date with respect to Calidria chrysotile?

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A. No, sir.
Q. Are you aware of such modeling he has done with respect to other types of chrysotile fiber?
A. Yes.
Q. Has that been published work?
A. Yes.
Q. Is there also unpublished work?
A. I assume.
Q. Is there any that you have seen?
A. No, sir.
Q. So what you have seen is his published work concerning modeling of chrysotile degradation or dissolution?
A. Yes.
Q. Do you recall offhand which chrysotile he used? First of all, was it all Canadian?
A. I don't recall whether it was Canadian or Rhodesian. I don't recall.
Q. That's kind of a small point, but let's put it this way, do you know if he has done -- when I earlier asked you whether he had done such modeling with respect to Coalinga, I wasn't limiting myself to Union Carbide Coalinga,

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I was limiting myself to the deposit.
A. Yeah, sure. Yes.
Q. So basically if I can summarize the request you have made of Rimstidt, it's that you would like him to model the dissolution or degradation of Coalinga type chrysotile and compare it with other types that he has modeled?
A. Yes, sir.
Q. Is that a request that you have made of Union Carbide, to do that work?
A. Not yet.
Q. Is that a request that you intend to make?
A. Yes, sir.
Q. Let me ask you the same question. Do you have any estimate of how long that work would take, if you get the go-ahead to do it?
A. No, sir.
Q. Have we now covered all of the original topic, laboratory work that you have requested with respect to Coalinga asbestos? Have we now covered it all?
A. Off the top of my head, yes.
Q. Have you requested any TEM analysis of

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Ilgren
the fiber itself?

A. Possibly surface microtopography and some other detailed analytical things.
Q. By TEM?
A. I don't know what the machine is. It's a form of electron microscopy.
Q. When you say possibly, have you actually made a request for some work or you can't remember if you did or not?
A. Request from whom?
Q. The original question was have you requested any TEM analysis of Calidria fiber, and you said you might have possibly requested some surface topography analysis.
A. I discussed some with Professor Frank Aplan at Penn State, and obviously Eric Chatfield in Ontario.
Q. So you think you may have discussed it with one or both of those?
A. Possibly others. I don't recall.
Q. Do you have any record of that?
A. Not to my knowledge.
Q. Has any such work been done?
A. No.

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Ilgren

Q. Do you have any anticipation that it will be done?
A. Perhaps.
Q. Have you gotten any go-ahead? Have you requested of Union Carbide that such work be done?
A. No.
Q. So you obviously haven't gotten any go-ahead from Union Carbide to do such work.
A. Yes.
Q. What question in particular are you looking at with respect to surface analysis of Calidria fiber?
A. What am I looking for?
Q. Right.
A. Anything unusual about the surface.
Q. Have you requested that any TEM or other microscopic analysis be done on Calidria with respect to size distribution of the fiber, either in an aerosol or in a wet solution or in any other form?
A. Yes, sir.
Q. Has that work been done?
A. No, sir.

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Ilgren

Q. To whom did you make that request?
A. Eric Chatfield.
Q. Have you asked Union Carbide for

authorization to do that work?
A. I think so.
Q. Do you know if that's been granted?
A. I believe so.
Q. Do you know if that work has begun?
A. It has not.
Q. Do you anticipate that it will begin within the next foreseeable future?
A. Hopefully.
Q. What specifically is that project? Is that size distribution in an aerosol or in lung tissue or in a solution or what?
A. All three.
Q. All three of those forms?
A. Yes, sir.
Q. Have you provided any lung tissue to Dr. Chatfield for purposes of doing that size distribution?
A. No, sir.
Q. Do you anticipate that he would use tissue from a Conwed worker or from some other

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Ilgren

source to do that?
A. Possibly both.
Q. Do you have available to you any lung tissue from any Calidria-exposed workers that can be used for that worker?
A. No, sir.
Q. So if that was to be done, you would have to obtain some tissue, would that be fair to say?
A. Yes.
Q. How would you go about obtaining that tissue, if you wanted to go ahead with that project?
A. Call Mr. Gerson.
Q. Have we now covered the requests for analytical work that you have made with respect to Calidria asbestos, or can you think of anything else?
A. At the moment I can't think of anything else.
Q. Let's go back to where we originally started this discussion, and that was with respect to the unpublished reports, papers or analyses you have seen with respect to Calidria

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Ilgren

asbestos.
Believe it or not, we started this discussion, we covered the Minnesota Department of Health and Dr. Weiss, and that's where we began, so let's go to the next name, Arthur

Morgan.
We have already talked a little bit about Dr. Morgan, but you indicated you have seen some sort of paper or report from him for something other than this dissolution work you have requested him to do. Do you recall what that was?
A. Specific to Calidria?
Q. Right.
A. I don't think I said -- was he among the unpublished?
Q. Right.
A. Take him out. He is not unpublished.
Q. You have seen unpublished work by Langer. What unpublished work have you seen by Langer? All these questions deal with Calidria asbestos.
A. His mineralogy report.
Q. The mineralogy report generated in

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Ilgren

connection with this case?
A. Yes. I think that's all.
Q. Have you seen any fiber burden analysis of tissue that Langer has generated in any workers exposed to Calidria?
A. No, sir.
Q. Have you seen any reports or unpublished work by Langer other than his mineralogical analysis in this case?
A. No.
Q. Next was Kevin Browne. What have you seen from him relating to Calidria?
A. You should take him out.
Q. You told us earlier about discussions you had with Kevin Browne.
A. Yes.
Q. But are you saying now that you can't recall any specific written reports?
A. No, there would be no reports.
Q. Or data from Browne.
A. There is someone I forgot, Chwastiak.
(Recess taken.)
BY MR. BROWNSON:
Q. Before we went off the record we had

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Ilgren

just stricken the name of Kevin Browne from the list of people who have provided you with unpublished reports or papers and you substituted a different name and I didn't catch it. What was that name?
A. I wrote it down. It's Chwastiak.
Q. I am not familiar with that. Who is

that? What's his first name?
A. Steven.
Q. Where is he located?
A. Presently?
Q. What's his current affiliation?
A. I don't know.
Q. Is he a medical doctor?
A. No.
Q. What is he?
A. I think he is an analytical chemist.
Q. What have you seen from Chwastiak?
A. Documents.
Q. Are these analytical work with respect to Calidria?
A. Yes, analytical work.
Q. Is it chemical analysis?
A. Some.

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Ilgren

Q. Does it deal with dissolution of Calidria?
A. Some.
Q. Degradation of Calidria?
A. Some.
Q. How about differential dissolution or degradation compared to other chrysotiles?
A. I think so.
Q. What form does this work take? Is it a report or tabular data or what is it?
A. I think it's just a UCC report.
Q. When you say a UCC report, it's a report prepared for UCC?
A. I believe so.
Q. Is this something that was provided to you by Mr. Gerson or did you get this from Chwastiak?
A. Gerson.
Q. Do you recall when this report was prepared?
A. I think 1963.
Q. The Chwastiak report, did you understand Chwastiak to be an employee of Union Carbide at the time he generated that report?

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A. Yes.
Q. So what you saw was a report circa 1963 generated by some sort of analytical chemist named Chwastiak concerning the Calidria fiber, would that be a description of what it is?
A. Yes.
Q. Was that a single report or was that a series of documents or what was that?
A. I don't recall.

Q. The next name you gave me was IITRI. What have you seen by them?
A. Analytical studies of Calidria chrysotile.
Q. Who is IITRI, what's the name?
A. Illinois Institute of Technology and Research Institute.
Q. So ITTRI is an acronym for Illinois Institute of Technology and Research or something along those lines?
A. Yes.
Q. When was this study prepared?
A. 1975 through 1984.
Q. Was this a series of analyses that were done over that time period?

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Ilgren

A. I believe so.
Q. What sort of analyses were these?
A. Physical and chemical analyses.
Q. Of fiber?
A. Yes.
Q. Was it fiber in any particular medium or circumstance or just fiber?
A. I think it would be air, water and animal feed.
Q. Animal feed?
A. Yes.
Q. Do you recall the context in which these studies were performed, why they were being done?
A. I believe it was at the request of NIESH.
Q. Were these provided to you by Mr. Gerson?
A. No.
Q. Where did you get that from?
A. From IITRI.
Q. How did you come to find out about them?
A. Indirectly through Hartwig Muhle.

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Q. So in your conversations with Hartwig Muhle, he indicated to you that the IITRI group had done some analytical work?
A. No. I asked him where he got his sample for inhalation testing from and he said NIESH, and I asked him for a copy of the NIESH report describing the sample used, and in the NIESH report it said that the material had been prepared by IITRI.
Q. Did you then go to IITRI and ask what material they had?

A. Yes.
Q. What they sent you was this series of analytical work done from 1975 to 1984?
A. Eventually, yes.
Q. What was this, was this microscopy or chemical analysis? What are we talking about here?
A. Microscopy, chemical analysis, XRD, a great number of different. I can't remember them all.
Q. XRD is x-ray diffraction?
A. Yes.
Q. Did this report take a narrative form

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or was it tabular data or what did it look like?
A. Both text and tables, graphs, figures.
Q. What you understood this analysis to be, then, was a sample or samples of Coalinga asbestos that was then used by Muhle for his aerosol generation?
A. One of the samples he had used, yes.
Q. Have you ever seen any other NIESH or documents or data concerning Coalinga asbestos?
A. I think so.
Q. Have you ever seen the NIESH air measurements of dust at the mill in King City, California done in about 1984 or thereabouts?
MR. GERSON: Objection to relevance. Go ahead.
A. I think so. I don't recall.
Q. I didn't catch your answer.
A. Yes, I believe so.
Q. Do you have a copy of that in your possession?
MR. GERSON: With him today or generally?
MR. BROWNSON: Not with him today, but in his possession in Bryn Mawr or wherever.

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A. I'm hesitating because I can't remember whether it was done, I think it was done by NIESH, but if it was and if I have it, it would be in Bryn Mawr.
Q. When you hesitate, let me ask some broader questions. Do you recall seeing air sampling data from the mill at King City, California from some source and you're just trying to remember if there was one from NIESH?
A. Exactly.
Q. Does some NIESH of about 1984 ring a bell and you can't pin it down?
A. Yes, sir.

Q. Other than that data and this IITRI data, to the extent that that's NIESH data, I guess it really isn't, is there other NIESH data you have seen concerning Coalinga fiber?
A. Not that I recall.
Q. Have you spoken to anyone at NIESH about Coalinga fiber?
A. Yes.
Q. Who is that?
A. I spoke to Dr. Jean Haartz. I spoke to Dr. Charles Lorboreau. I think they are the

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only two. There may be one or two others.
Q. Are they both at NIESH in Cincinnati?
A. Yes, I believe they are in Cincinnati.
Q. Do you know when you spoke to them? Was that recently or when?
A. Last year sometime.
Q. Was that done in connection with this case or for other purposes?
A. Probably both.
Q. Did that generate any sort of written correspondence or notes or documents?
A. Some small correspondence.
Q. That would be correspondence back and forth between you and those two NIESH doctors?
A. Yes.
Q. Do you still maintain copies of that correspondence?
A. I don't know.
Q. If you did, would they be in your materials at Bryn Mawr?
A. Yes.
Q. What was the reason for your inquiry to those doctors? What was the question or the topic that you posed to them?

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A. It was my understanding that IITRI prepared samples for NIESH of both Calidria and non-Calidria chrysotile types that would serve as worldwide standards, and I wanted to track down anyone in the world who had ever done any research on Calidria, so I wanted to try to get a listing from them of everyone in the world to whom they had sent samples.

I also wanted to try to clarify exactly what kind of Calidria preparation they had been using.

Q. First of all, are those two men medical doctors or are they industrial hygienists, or who are they?
A. I think Jean is a woman, but that's

not important.
Q. Those two individuals?
A. Yes, individuals; and I think they are both Ph.D.'s.
Q. What answer did you get to your inquiry to them about who had standardized samples of Calidria fiber for analysis?
A. What answer?
Q. What did you learn? First of all, had

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they done that?
A. Had they done what?
Q. Had they prepared some standard preparations of Calidria?
A. Yes.
Q. Was that done again for NIESH or for who?
A. I don't know why they did it. I guess that's just what they do.
Q. As long as we are on that topic, I have a paper which you have probably seen entitled "Characterization Of Three Types Of Chrysotile Asbestos After Aerosolization" published in Environmental Research in 1983 by Pinkerton, Arnold, Brody and others.
Are you familiar with that paper?
A. Yes.
Q. In that paper it is said that what they were trying to do was prepare some standard preparations of Coalinga fiber and some fiber from the Jeffrey pit in Quebec for use in experimental research by the National Institute of Environmental Health Sciences.
A. That's not exactly correct. I don't

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think they were preparing standards. I think they were just characterizing potential standards. It's semantical.
Q. I guess it is kind of semantical, but as you understand it, they were trying to characterize samples, some standard samples that had been prepared of these two types of fiber?
A. I think they are one and the same.
Q. I think we are getting into a semantic argument here, but when you were talking about these two Ph.D.'s at NIESH, as you understand it they were doing the same thing, trying to get some standard samples for --
A. I think they just served as a respository so that if one wants to do some Calidria research, research on Calidria fiber, you call them up. I don't know how one becomes

aware that they serve as a respository, but say you want a gram or whatever of material to do a study with, you just call them up and say I would like to do this.

I guess by analogy to the UICC standard samples, they probably serve as a respository. They had something like 35

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different types of chrysotile and half a dozen types of amosite and other fiber types, so this one was CH-29, this one being the Calidria, but they had I think CH-30 was Arizona and CH-27 was something else, so I don't think there was any type of competition.

In fact I think the sort of life cycle chain went something like NIESH requests that IITRI serve as outside contractor to characterize a sample of Calidria fiber. IITRI contacts Union Carbide, requests a sample, Union Carbide sends IITRI material for analysis, IITRI analyzes the sample.

It would then go back to NIESH within the respository, and then NIESH -- i.e., Pinkerton, et cetera -- may wish to use that, or the 25 other groups through the world who made subsequent requests after the respository was set up.

I think that's the way it works.

Q. So, for instance, if you wanted to do some further characterization of Calidria samples, like let's say this project you were talking about with Muhle, is that where you

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anticipate you would go to get samples, that respository at NIESH in Cincinnati?

A. Perhaps.

Q. Let me ask a different question.

Would samples be available at that respository if you requested them for work of that type?

A. I believe so.

Q. Let me ask one more name. The next name you gave us was Martin. What Martin is that?

A. I don't think that's unpublished, I'm sorry. I was mixing there. I think he is published, so let's take him off the unpublished list.

Q. Let's put him on the published list. Which Martin is that and what has he published?

A. I think his first name is Colin, but I can't remember. I think it was something like

"Thermographic Aspects Of Chrysotile Asbestos," Mineral Magazine 1977.
Q. Do you understand that one of the types of chrysotile that was included in that thermographic analysis was Coalinga?

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A. Yes.
Q. You say that was published in Mineral Magazine in approximately 1977?
A. I think so.
Q. Who is Colin Martin, is he a mineralogist?
A. I think so.
Q. Where is he located or was he at the time he published?
A. Oxford.
Q. How did you come to find out about that publication?
A. From the literature.
Q. My search of the literature didn't turn that up, using databases I had. I'm just curious what search you made that turned up that reference.

(Continued on the following page.)

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A. I can't recall. I don't recall.
Q. But in any event it turned up and it was published, as you recall, and not unpublished, is that fair to say?
A. That's correct.
MR. GERSON: Why don't we take a break.
(Luncheon recess taken at 12:40 p.m.)

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AFTERNOON SESSION

(2:20 p.m.)

EDWARD B. ILGREN,

resumed, having been previously duly sworn, was examined and testified further as follows:

EXAMINATION (Cont'd.)

BY MR. BROWNSON:

Q. When we broke for lunch we were going through the list of individuals you had named from whom you have unpublished material dealing with Coalinga fiber.

The next name we had on the list is that of Jean Haartz. You had earlier told us about a Dr. Haartz at NIESH. Is this the same one?

A. Yes.

Q. The published reports you spoke of earlier from this Dr. Haartz, are those the same standardized preparation reports from NIESH that you mentioned earlier or is it something else?

A. The same.

Q. Next was Graf.

A. Right.

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Q. Who was that?

A. She was the research chemist analyst at IITRI who did a lot of the hands-on research.

Q. Would the reports where her name appears be the same ones you mentioned earlier, the IITRI studies?

A. Right.

Q. Next you mentioned Mumpton, Fred Mumpton?

A. Yes.

Q. Do you know what papers or reports you have from Fred Mumpton?

A. Again, I think I have a Union Carbide report.

Q. Do you recall that this was one or a series of reports he did back in the mid-'60s where he was analyzing the ore from the deposit, or is it something other than that?

A. It would be that.

Q. Where did you obtain the Mumpton reports, do you know?

A. Either from Virginia or Alan.

Q. I don't mean to quibble over this

because it's a small point, but Mumpton has two papers that I didn't bring with me that actually saw the light of day in journals.

I think they are the same ones you are talking about, but do you recall them being published anywhere or do they appear to you to be unpublished?

A. They appear unpublished.

Q. Have you seen any published work by Mumpton?

A. Yes.

Q. So this is something other than work you have seen published in journals?

A. Yes.

Q. Again, do you recall how many reports it is? Is it a couple?

A. It's at least one.

Q. If you can recall, does that report have in it photographs, I guess it wouldn't be an original photograph, but a photo of, actually I guess it would be a photomicrograph under the electron microscope of the Calidria fiber?

A. I can't recall.

Q. Do you recall seeing such photos that Mumpton took in the '60s, whether in the published papers or in those reports?

A. I certainly believe there is a TEM shot in the published papers. I don't recall in this unpublished document.

Q. Do you have any information as to the date or the approximate time frame when Union Carbide had available in its own laboratories a transmission electron microscope?

A. When did they first buy a scope?

MR. WILL: An electron microscope.

A. No idea.

Q. The next name you mentioned was Thickstun. Who was that?

A. I think Thickstun was one of the geologists that worked either independently or with Condor between 1950 and 1957 on the New Idria serpentinite. on the body, and he was the one who met Bill Cohan and Ary. Cohan was the chief geologist for UCC and Ary was the vice president at UCC Nuclear up in Reno, and Thickstun was the one in this kind of semi-anecdotal account, entitled something like Hell's Lid, Asbestos As Hell's Lid, a funny title, but he was just a geologist who describes

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the deposit.
Q. Does it appear to be a description based on his own analysis of things, or is it kind of a historical perspective of this?
A. It's a mixture of historical analysis, some of his own or his colleagues' analyses. It refers to different samples that he had analyzed because some people say it was mountain leather or an amphibole, but then the report came back that it was only chrysotile, so there is a mixture of historical description, personal analysis and allusion to analyses done by the Department of Mineralogy in California early on in the early '50s.
Q. Then you also mentioned Bright. Who was that?
A. Bright again was another geologist who wrote this document in 1959, and I can't remember if it's a UCC document or what.
Q. So at any rate you got it through Mr. Gerson's office, but you don't know if it was actually prepared by UCC originally?
A. That's correct.
Q. Do you know who Bright was affiliated

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with at the time he wrote that report?
A. I don't recall.
Q. Next you mentioned Addison.
A. Yes.
Q. Which Addison is that?
A. John.
Q. What do you have from John Addison?
A. Just a letter.
Q. Is this a letter addressed to you or someone else?
A. To me.
Q. What's the topic of the letter?
A. Just a small discussion of I think Yeager's '83.
Q. Is this a paper by Yeager and Kagan and other authors dealing with dosing human macrophages with Calidria and other fibers?
A. Yes.
Q. Would it be fair to say that what you have is a letter from John Addison written to you commenting on that paper by Yeager?
A. Yes.
Q. What occasioned Dr. Addison to write you about the paper? Had you sought a critique

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from him or did he send this out unsolicited or

how did that come into your possession?
A. I asked him to critique a certain part of the minerological statement, if I recall, the fiber analysis aspect.
Q. Do you recall what you asked him to critique specifically?
A. Not at the moment.
Q. Do you recall what his critique said, what the letter says?
A. I have reviewed a lot. I would have to have it before me.
Q. Next you said Hodgson?
A. Yes.
Q. What Hodgson is that?
A. That's Alan A. Hodgson.
Q. What do you have from Hodgson?
A. He wrote a small report on sort of his thoughts on Calidria fiber.
Q. When you say a small report, is it in the form of a letter?
A. Yes, a letter.
Q. It's a letter addressed to whom?
A. I think it's to me or for me.

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Q. Is it a letter in any event that you asked be written by Dr. Hodgson?
A. I think he just said he would put some thoughts down.
Q. Was this in response to an inquiry from you?
A. We had just been discussing Calidria fiber, what might be peculiar about it.
Q. You said could you please put some of your thoughts down and he followed that up with writing a letter where he put some of his thoughts down?
MR. WILL: Objection. I think he previously testified it was Alan Hodgson would put some thoughts down.
MR. BROWNSON: I don't follow you there.
MR. WILL: You suggested that Mr. Ilgren had proposed that this be reduced to writing. The earlier answer was that Dr. Hodgson had suggested that.
THE WITNESS: That's correct.
Q. So let's see if I can get the sequence straight. At some point you and Dr. Hodgson were

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sitting around discussing Calidria asbestos?
A. Yes.
Q. Would this discussion have occurred

after you were first hired by Union Carbide to look into aspects of Calidria or before that time?
A. Probably before. We might have first talked in '87, '88, a long time ago.
Q. How was it, as best you can recall, that the subject of Calidria fiber came up at that conversation?
A. In the context of slip fiber.
Q. Did this conversation arise in the context where you had some sort of scientific meeting or assembly or what?
A. I was asked by Celotex to research the potential biological effects of slip versus cross fiber in '86.
Q. Was that in connection with fiber from Carey Canada, the Celotex subsidiary?
A. Yes.
Q. I assume that Carey was mining some slip fiber in Canada?
A. It is slip fiber.

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Q. Do you know what mine or pit that was coming out of?
A. Pennington Dike, East Broughton.
Q. So if we can finish the topic of the Hodgson discussion, you were doing some investigation for Celotex in connection with the Carey Canada slip fiber in about '87?
A. Yes.
Q. Do you know who requested you to do that investigation?
A. I think the request came from some senior person at Celotex.
Q. Do you know if it came from one of the lawyers at the Montgomery McCracken firm in Philadelphia?
A. It came through Phil Vogler, I think.
Q. As you can recall, and I know it's going back seven years, was that in connection with some of the asbestos building litigation that Phil was working on for Celotex at that time?
A. No, I don't think it was specific to any building litigation. It was a general inquiry because their deposit was largely slip.

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Q. In any event, in conducting that investigation did you contact Dr. Hodgson?
A. Yes.
Q. The two of you then met?
A. Yes.

Q. He then followed up the meeting with a letter to you?
A. This is years ago. I mean we have been friends and colleagues for a long time. Over the last six or seven months or in fact the last couple of years I have been talking to him off and on about Calidria fiber.
Q. This particular letter that you mentioned that got this discussion going, I understood that letter was dated at or around 1987.
A. No, that letter would be quite recent, a few months ago.
Q. The next name you gave me was Jordan.
A. Yes.
Q. What Jordan is that?
A. I think he is either an analytical chemist or an engineer, a UCC employee.
Q. What report or documents do you have

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from him?
A. Commercial properties of Calidria fiber.
Q. There were a series of papers put out by UCC people, including Jordan, dealing with the properties of Calidria fiber in three basic industries. One was paper-making, one was plastics and one, I believe, could have been floor tile, but I know one was drilling mud for the oil drilling industry.
Do you recall in which context this paper appeared?
A. I haven't the details of the papers. I don't recall drilling mud. I don't recall any specific. I think these were more theoretical papers.
Q. Let me narrow it down.
Do you recall the Jordan paper as dealing with the commercial properties of Calidria asbestos for use in paper-making?
A. I believe so. It's more of a guess.
MR. WILL: Don't guess.
Q. Do you recall the date of that paper?
A. Mid-'60s.

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Q. The next name is Park. Who is that?
A. I think his name is Kisoon Park, another UCC scientist working on Calidria fiber for quite some time.
Q. This was another of this series of Union Carbide papers dealing with Calidria fiber?
A. Yes.

Q. Then you mentioned Woolery, Robert Woolery?
A. Yes.
Q. What do you have from him?
A. Again other papers describing commercial applications.
Q. Morgan in particular wrote a paper or authored a paper called "The Use of Coalinga in the Paper-Making Process." Do you have a copy of that?
A. Yes.
Q. Are there others that you can recall other than that?
A. Not by detailed title, but I do recall others.
Q. Would it be fair to say that you have more than one Woolery paper but you don't know

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how many?
A. Unpublished or published?
Q. Unpublished.
A. I think I have more than one.
Q. Do you have any published material by Woolery?
A. Yes.
Q. Do you recall what the published papers are that you have?
A. I think one is the TAPPI paper. I think that's Technical Applications of the Paper and Pulp Institute, whatever. The other journals don't immediately spring to mind.
Q. Next you mentioned Dresher.
A. Yes.
Q. What is that? What do you have?
A. Who is he?
Q. Okay, who is Dresher?
A. Dresher again was an analytical chemist who worked for UCC.
Q. Do you have a paper that he authored?
A. Yes.
Q. Is there more than one or just one?
A. I believe there is more than one.

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Q. Do you recall if any of those papers deal with the properties or the use of Coalinga fiber in the paper-making process?
A. I believe so, but I'm not sure.
Q. Then the final name you gave us is Nauman.
A. Yes.
Q. What do you have from Nauman?
A. He worked with Dresher. Again I have

some unpublished papers.
Q. Let me turn to a different topic. That's your CV. Let's mark a copy of a CV which Mr. Will provided to me about a month ago, recently.
(Ilgren Exhibit 1, Ilgren CV, marked for identification, as of this date.)
(Ilgren Exhibit 2, Ilgren CV, marked for identification, as of this date.)
(Ilgren Exhibit 3, Ilgren CV, marked for identification, as of this date.)
Q. Let's look at Exhibit 1. We have marked as Exhibit 1 a curriculum vitae that has a date stamp of May 5, 1994 on it, which is an undated CV.

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Is this an up-to-date CV?
A. It looks fairly up to date.
Q. This date stamp on the front, I assume that's a date stamp, it's not one from my office but maybe from Mr. Will's office?
MR. WILL: No, it's not from my office.
Q. Do you know where that came from?
A. No.
Q. Would it be fair to say that this is up to date at least as of May 5, 1994?
A. Yes.
Q. Is there anything since that time that we should add to this or change?
A. I would have to go through it in detail. Not off the top of my head, no.
Q. I would like to go through a few things on the CV in Exhibit 1.
First of all, is this a CV that you prepared yourself?
A. Yes.
Q. It indicates that you have an M.A. from Oxford?
A. Yes.

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Q. An M.D. from where?
A. Hahnemann.
Q. Then it says D.Phil. from Oxford?
A. Yes.
Q. Then MACPath.?
A. American Board of Pathology.
Q. And the last reference is MRCPath.?
A. Royal College, sort of the English equivalent to boards in neuropathology.
Q. What does MRCPath. stand for?
A. Member of the Royal College of

Pathology.
Q. On the second page of the CV there is the heading "Education."
The first category is "Institute and Location," and then it says in the second column "Degree and Field of Study," and third is "Year Confirmed." Do you see that?
A. Yes.
Q. That indicates you got an M.D. from Hahnemann in 1974, is that correct?
A. Right.
Q. Then the second thing on there is American College of Pathology.

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A. That would be Board certification.
Q. Then if you look at degree and field of study under that one, it says MACPath. Does that stand for Member, American College of Pathology?
A. In England, this CV was written largely for English people, so they don't have boards of some specialty. They have colleges of specialties. Instead of getting the boards, you get a membership, so I was trying to Anglicize this for people to understand in Europe and in England rather than America.
Q. So what this reference refers to is that you're Board-certified in pathology in the United States?
A. Yes.
Q. It doesn't refer to the fact that you're a member of the American College of Pathologists?
A. Yes, that's true.
Q. Was my statement correct, you're not a member of the American College of Pathologists?
A. I guess it's a diplomate of the American Board of Pathology.

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Q. I'm not trying to make a big distinction here, but in this country in the United States you can be Board-certified in pathology, which is something that comes from, actually I guess it's down in Tampa or someplace, the Board certification.
A. Chicago.
Q. But then there also is an American College of Pathologists?
A. Really?
Q. I guess what I'm getting is what this indicates is you're Board-certified?
A. Correct.

Q. It's not an indication that you're a member of the American College of Pathologists?
A. I didn't even know there was a College of Pathologists.
Q. It indicates here that the Institute is the American College of Pathology.
A. I should correct that. The American boards.
Q. The next is University of Oxford, D.Phil. from Oxford University in England?
A. Yes.

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Q. 1980?
A. Yes.
Q. That was in the field of zoology/biology?
A. Yes.
Q. I'm going to jump ahead, but the last entry, University of Oxford M.A.?
A. Yes.
Q. In biology and medicine, 1983?
A. Yes.
Q. My question is how come you got the D.Phil., which I shouldn't call a Ph.D., before the M.A.?
A. It's an M.A. that's conferred, it's not a thesis M.A. where you write a treatise or something. It's a degree which they give you as a senior member of the university so that you can vote in university matters so you can be a thesis adviser. It's not equivalent to going to Columbia to do a master's in art history or something.
Q. So would it be fair to say that that is a degree from Oxford in 1983 that was not as a result of a specific dissertation or course of

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study that you took?
A. Yes.
Q. It was like a conferred degree of some sort?
A. Yes.
Q. Then the next entry on there is Royal College of Pathology.
A. Yes.
Q. 1982?
A. Yes.
Q. It says under degree and field of study, MRCPath. neuropathology. What does that stand for?
A. That's the Royal College of Pathologists membership, which again you sit down

and take your exam and look at slides. It's the same as the American boards of neuropathology.
Q. That was obtained in 1982?
A. Yes.
Q. It indicates degree. That's not actually a degree, is it? Isn't that, it's similar to American Board-certification in pathology?
A. My understanding in England is that's

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a degree. In this country it wouldn't be a degree. I guess it's definitional, sort of.
Q. Did you earn any degrees since 1983?
A. No.
Q. Would it be fair to say that the last earned degree would be the D.Phil. you got from Oxford in 1980?
A. By American standards.
Q. Up until that time, had you treated patients with any asbestos-related disease?
A. Say that again.
Q. Up until 1980, had you treated patients with asbestos-related disease?
A. Treatment in the broadest sense of having looked at any slides from asbestos-related disease cases or whatever?
Q. Let's back up. I understand that you received your medical degree in 1984 and the degree was --
A. 1974.
Q. At that time, that was what's known as an M.D., a medical degree, correct?
A. Yes.
Q. Was it in any particular specialty at

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that time?
A. You don't specialize in that.
Q. Let's start there. My point is as of the time that you got out of Hahnemann, received your degree in '74 of M.D., up until that time in your medical school training had you ever actually treated patients with any asbestos-related disease?
A. Had I ever seen anybody with an asbestos-related disease in medical school?
Q. Right.
A. Probably, but I don't recall.
Q. Then after 1974 and until 1980 you were involved in various residencies or programs of one sort or another in this country and in England and you got this D.Phil. degree in zoology and botany in 1980, would that be a

thumbnail summary for that time period?
A. Yes.
Q. Would it be fair to say that what you were studying during that time period was neuropathology or not?
A. 1974 to '80, not at all.
Q. So up until 1980 you were not involved

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in the field of neuropathology?
A. No, except for maybe two months of my residency.
Q. At what point in the residency and where were you involved in neuropathology?
A. I did my residency at Cornell here in the city and as part of the anatomic pathology residency you do two months of neuropathology, two months of pediatric pathology, ten months of surgical pathology, eight months of autopsy pathology. I can't remember. A month or two of psychological pathology.
Q. This resume indicates that you are a member of the faculty of biological and agricultural sciences at University of Oxford?
A. That's correct.
Q. Is that true today?
A. Yes.
Q. But you don't live in England at the present time?
A. No.
Q. Where do you currently reside?
A. Bryn Mawr, Pennsylvania.
Q. Is that your office location?

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A. Office and home.
Q. What's your office address?
A. Department 503, 830 Montgomery Avenue, Bryn Mawr, Pennsylvania.
Q. How long have you had your office at that address at Bryn Mawr?
A. About two years now.
Q. Do you still maintain an office in Oxford?
A. No.
Q. How is it, I'm not up to date on the English system, but how is it you're still on the faculty at Oxford when you haven't lived there for two years?
A. It's a more liberal system. I mean it's not an appointment where I have to do a set number of teaching hours or administration or anything of that sort. It's more a question of just continuing on with research and writing. I

had planned to go back next year. I suppose there was a question of going back, so it makes sense.
Q. Is there a certain time period that you can be away from the University of Oxford and
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still be on the faculty?
A. I would imagine five or six years.
Q. In other words they haven't said to you get back here by next year or we are going to kick you off the faculty?
A. No.
Q. The faculty that you're on, as I understand it, is the biological and agricultural sciences?
A. Yes.
Q. Is that a particular school or college at Oxford?
A. I guess you could compare it to the school of dentistry or the school of medicine or the school of biology or whatever.
Q. Is there any separate and distinct school of medicine at Oxford?
A. Yes.
Q. So this faculty, then, is not in the school of medicine?
A. No.
Q. It's in a different school?
A. Yes.
Q. Is St. Edmund Hall a specific school
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at Oxford?
A. That's one of the 38 colleges at the university.
Q. Is the faculty of biological and agricultural sciences only within St. Edmund Hall or does it cross within several of those colleges?
A. It crosses within all colleges.
Q. So see if I have this straight. You're a member of St. Edmund Hall?
A. Correct.
Q. What specifically does it mean when they say you are a member of one of the colleges at Oxford?
A. You have to be a member of a college to be a member of the university. That's just, I suppose, a formality. It's your college. They are separate. The college system to some extent is separate from the faculty.
Q. So when you say it's your college, that's the college where you received your

degree, for example?
A. Yes.
Q. Was St. Edmund Hall?

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A. Yes.
Q. In describing your current faculty position, would it be fair to say you're on the faculty of St. Edmund Hall?
A. No.
Q. You're on the faculty of biological and agricultural sciences?
A. Yes.
Q. And you're affiliated, then, with the college of St. Edmund Hall?
A. Yes.
Q. Do you have a term or is there a word or description that they use to describe that affiliation with St. Edmund Hall?
A. Just a research fellow or research scientist.
Q. Do you have any laboratory or research facilities at St. Edmund Hall College?
A. No.
Q. Do they pay you any sort of faculty salary there?
A. No.
Q. So when they say you're a research fellow or research scientist, that's a voluntary

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position?
A. More or less, yes.
Q. Do you have to pay anything like tuition to maintain that position?
A. No.
Q. You're just there. They don't pay you, you don't pay them, you just hold that position?
A. Yes.
Q. Are you required to do any certain work to maintain that position at St. Edmund Hall?
A. No.
Q. Do you do any teaching at the college of St. Edmund Hall?
A. Not at the moment.
Q. When is the last time you did any teaching at that college. St. Edmund Hall?
A. University teaching between 1980 and 1985 was the last time I did formal teaching.
Q. At St. Edmund Hall or anywhere at Oxford?
A. It would be at the University Medical

School.

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Q. Oxford University Medical School?
A. Yes.
Q. Are you required to do any research to be on the faculty of St. Edmund Hall?
A. No.
Q. How about to be on the faculty of biological and agricultural sciences?
A. That's the principal duty, just to do your research.
Q. Here you hear the term or phrase "publish or perish" in American colleges. Is there such a concept at Oxford, where you are required to do so much publication?
A. Obviously they like you to keep publishing. I would say that's important.
Q. So is there any particular requirement or is it kind of an unspoken rule?
A. I suppose it's unspoken.
Q. You say you're supposed to continue to do your research. What research are you currently doing as a member of the faculty of biological and agricultural sciences?
A. I look into fiber type differences for different forms of asbestos, different forms of

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zeolite. The research is indicated in the CV, page -- the research experience is outlined on page 6, 7 and 8 and the sorts of things I have been doing lately are outlined at the end of page 8-I.

MR. BROWNSON: Let's mark this as Exhibit 4.

(Ilgren Exhibit 4, letter dated 25 April 1994, Moss to "Dear Sir or Madam," marked for identification, as of this date.)

Q. We have marked as Exhibit 4 a letter that I received or actually I had my paralegal receive because I'm unfamiliar with the system at Oxford. I made an inquiry as to your faculty position and I received this letter of April 25, 1994 from P.W. Moss.

Do you know who that is?

A. I have no idea.
Q. It indicates he is the head clerk. Do you know what that position is?
A. I guess it's one of those antiquated titles that they have in the administration.
Q. Here it says Dr. Ilgren, paraphrasing, was a member of the university staff from 1984 to

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1987 when he left.
Do you know why it is that the head clerk at Oxford would indicate that you left in 1987?
MR. GERSON: Left what?
MR. BROWNSON: Oxford.
MR. WILL: It doesn't say that in the letter.
MR. GERSON: Why he left the university staff specifically?
MR. BROWNSON: Right. It says he was a member of the university staff from 1984 to 1987, when he left.
Q. Did you leave the university staff at Oxford in 1987?
A. It's news to me.
Q. Then he says "I do not have a forwarding address."
Is it your testimony that at all times since 1987 you have had an address of some sort at Oxford?
A. Well, I have gotten mail from St. Edmund Hall, so I assume, and I have sent letters to St. Edmund Hall.

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Q. Have you actually had an office at Oxford since 1987 at any time?
A. I was working with the Imperial Cancer Research Fund and I assume that was my base until at least 1989. I would have to go and check the dates.
Q. Is the Imperial Cancer Research Fund a school or college of Oxford or is that something else?
A. It's in the Nuffield Department of Clinical Medicine. It was under Richard Peto.
Q. So it's your testimony that since 1987 you have had an office address at the Imperial Cancer Research Fund in that department at Oxford?
A. Up until I would say 1989. Again, I would have to check the dates.
Q. Then he goes on to say "He was a member of St. Edmund Hall within the University. I suggest you contact the College Office for possible further information."
I read this to mean that as of April 24, 1994 you were no longer a member of St. Edmund Hall, is that a fair statement?

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MR. WILL: Are you asking him is that

what he thinks Moss is saying or are you asking him whether it's a fact that he was no longer a member of the university staff as of '87?

Q. I will rephrase the question. As of April 25, 1994, were you a member of St. Edmund Hall within the university?

A. I believe so, but as I say, I didn't know any differently.

Q. When you say you believe so, did you actually have a physical office with a desk and chair and that sort of thing?

A. No.

Q. Did you have like a post office box or what would it be?

A. Just a member of the college. If you're not living there, you don't keep a post box or mailbox.

Q. So is it your testimony that you were a member of the College of St. Edmund Hall as of April 25, 1994 but you weren't physically present there?

A. Yes.

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Q. You were in Bryn Mawr, Pennsylvania?

A. Yes.

Q. I really don't understand this Oxford system, but is it fair to say or is it your testimony that you are currently a member of the staff at St. Edmund Hall?

A. The college staff, again accepting that this is a different system, but the college staff I would interpret as ten or twelve so-called fellows or dons that would have the principal teaching responsibilities at the college. They would give tutorials or lectures. So I would not be college staff.

Q. So you're not one of those people who teach on the staff of the college.

A. No.

Q. You never have been?

A. No.

Q. Do you currently do any teaching in the medical school at Oxford?

A. No.

Q. Have you ever done teaching in the medical school at Oxford?

A. Yes.

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Q. When was the last time you did that teaching?

A. I think I said the teaching was done

between 1980 and '85.
Q. Since that time have you presented any lectures in the medical school at Oxford?
A. No.
Q. In your current position you testified you're on the faculty of biological science. I have seen that expressed both as biological science and biological and agricultural science. Do you know which one it actually is?
A. Perhaps they dropped. It used to be BAS. I think now it's just biological science.
Q. In that capacity are you actually a fellow or a don, as you have described it?
A. No.
Q. Do you have an actual title in terms of what you are? Do they call you something?
A. No.
Q. You just say you're on the faculty?
A. Yes.
Q. Are you a senior lecturer?
A. No.

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Q. Have you ever been a senior lecturer?
A. I was a senior registrar who gave lectures, but the title of senior lecturer is a very specific title.
There were times when people might refer to me as a senior lecturer, but the neuropathology position was senior registrar. I gave lectures and sometimes one could be termed a senior lecturer.
Q. There actually is a title there called senior lecturer, correct?
A. I believe so.
Q. My question is, I guess it's a simple question, have you actually held that faculty title of senior lecturer?
A. I don't believe so.
(Ilgren Exhibit 5, excerpt from Official ABMS Directory of Board-Certified Medical Specialists 1994, marked for identification, as of this date.)
Q. I show you Exhibit 5. This is a photocopy of a title page and a reference from the Official ABHS Directory of Board-Certified Medical Specialists 1994.

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Are you familiar with that particular work or that reference guide?
A. Not really.
Q. This is a reference to American Board-certified specialists. Have you ever seen

that before that you can recall?
A. Something similar, probably.
Q. What I have attached is the page where your reference appears in the 1994 edition, which is the 26th edition.
It states that current hospital appointment is Radcliffe Infirmary at Oxford, United Kingdom. Was that true in 1994?
A. No.
Q. Then it says senior lecturer, University of Oxford. Was that true in 1994?
A. No.
Q. Then under that there is some designations. The first one is BSN. Do you know what that stands for?
A. I think it's British Society of Neuroscientists.
Q. Is that a society that you're a member of, was or are?

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A. British Society of Neuropathologists, yes, I was a member of that.
MR. GERSON: Let's take a two-minute break.
(Recess taken.)
BY MR. BROWNSON:
Q. We were looking at Exhibit 5. Again looking at this entry, and I'm trying to interpret what this means, the first entry there is Certificate Path. AP '77. Do you know what that refers to?
A. That's Board certification in pathology, 1977.
Q. As I look at Exhibit 1, which is the CV, it indicates you were Board-certified in 1976. Do you know which one is correct?
A. I would have to go back and check. It was the end of '76 or '77.
Q. So sitting here today, you don't know if you received your Board certification in pathology in 1976 or 1977?
A. It might be '77. I would have to go back and check.
Q. But you don't know which one it is

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without checking?
A. No, I'll check.
(Ilgren Exhibit 6, fax, St. Edmund Hall to Waskosky, marked for identification, as of this date.)
Q. I show you Exhibit 6, a letter addressed to Renee Waskosky, my paralegal, from

Mr. Gosling, principal, St. Edmund Hall, Oxford.
A. I just got a letter from him yesterday.
Q. As you can see, this was in response to a followup from the previous exhibit where it said inquire of St. Edmund's Hall.
Gosling writes -- first of all can you read what he wrote? Have you read it?
A. Yes.
Q. Is that all correct, what he says there?
A. It's correct. I thought it was that I am a member, but it looks like the membership lapsed.
Q. So if Gosling claims that as of April 28, 1994 you are not a member of the faculty of biological sciences, would that be

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news to you?
A. Yes, I believe so.
(Ilgren Exhibit 7, letter dated 8 April 1994, Roberts to Waskosky, marked for identification, as of this date.)
Q. Would you look at Exhibit 7. This is a faxed letter of April 25, 1994 to my paralegal, Renee Waskosky, from Professor Roberts, Registrar, Royal College of Pathologists in London, Patron, Her Majesty, the Queen.
Specifically I want to draw your attention to the second paragraph where it indicates that you're an associate member of the Royal College of Pathologists. Is that a correct statement?
A. Right, that's what it says here on the front of the CV.
Q. Then it says you gained exemption in October 1981 from the first part of the membership examination, the primary, is that correct?
A. Yes.
Q. It says the exemption was granted on the basis of your publications in the

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pathological field.
A. Yes.
Q. Do you know what those publications were that allowed you to get the exemption from the primary examination?
A. The first 25 or 30 papers that I wrote.
Q. So would it be fair to say it would be those papers which would show up on your CV as

published papers as of 1981?
A. It would have to be.
Q. I'm looking at the list of publications on your CV which is Exhibit 1, and that appears at page 12. I see that before October of '81, and the dates aren't entirely clear, there could be or there are, it appears, nine papers, references 1 through 9, is that correct?
A. Yes.
Q. Do any of those papers deal with the issue of asbestos or, deal with any issues pertaining to asbestos?
A. No.
Q. Going back to Exhibit 7, which is the

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letter from Professor C. Roberts, do you know who that is?
A. No.
Q. It says "The status of Associate is entirely optional on the part of the applicant. It does not permit entitlement to designated initials such as ARCPath. but does allow the holder to receive the quarterly mailings of the College."
A. I think that should be MRCPath.
Q. Do you agree with that statement?
A. Again, that's news to me, but I would be happy to drop it.
Q. I notice that that designation does appear on the cover page of your CV, correct?
A. Right, as associate in neuropathology.
Q. So would it be your testimony that at various times since October of 1981 you have used the designated initials MRCPath. on various CV's and designations and publications?
A. Sure.
(Ilgren Exhibit 8. excerpt from Directory of Royal College of Pathologists, marked for identification, as of this date.)

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Q. We have marked as Exhibit 8 a photocopy I have taken of the cover and an entry from the Royal College of Pathologists Directory of 1993. It lists your entry as Dr. E.B. Ilgren and then it lists last known address at care of Harvard Club, New York City, New York, New York. Would it be fair to say that as of that date you had no address at Oxford. at least with the Royal College of Pathologists?
A. Yes, I imagine. I don't know how the Harvard Club of New York got here.

Q. Do you know how come they wouldn't have been at your office in Bryn Mawr?
A. They just sent me a bill last week for annual membership, so I'm not quite sure why not. I'll be sure to tell them.
MR. WILL: The billing office has the right address.
Q. Did you ever during the time period 1993 or before reside at the Harvard Club of New York City?
A. Yes.
Q. When was that?
A. I must have been there for two and a

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half, three months in 1991 sometime.
Q. Were you actively engaged in any research projects while you were living at the Harvard Club of New York?
A. I was doing research with or for W.R. Grace. Actually physically having a lab in the club, are you asking?
Q. Let me put the question this way. At the time you were residing at the Harvard Club of New York, my question is what, if any, research were you engaged in during that time period?
A. I was going through documents concerning vermiculite and tremolite for W.R. Grace and documents and animal studies for Carborundum Corporation, ceramic fiber material.
I can't remember all the research I was doing at the time.
Q. Would it be fair to say you didn't have any laboratory facilities or office facilities at the Harvard Club?
A. Yes, that's true.
Q. Since 1985 has there been any office or laboratory of any kind at any college or department at Oxford that has been yours?

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A. Personally mine?
Q. Right. In other words where it would say on the door Dr. E.B. Ilgren?
A. No.
Q. When did you actually physically move your address from England to the United States?
A. I sold my house, which was at 13 Frenchay Road, Oxford, trying to think of when the completion date was. I think it was 1992 when it was officially sold.
Q. Since that time have you had any office or laboratory facilities other than the apartment in Bryn Mawr in the United States?

A. No.
Q. Would you agree with me that the principal organization of Board-certified pathologists in the United States is the College of American Pathologists?
A. You mean the boards of --
Q. Let me back up.
A. We went through this business about boards.
Q. I understand, but this is a different question. Let me back up and rephrase it.

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Would you agree that the principal organization of Board-certified pathologists in the United States is the College of American Pathologists?
A. I don't understand it.
MR. WILL: I don't either.
(The record was read.)
MR. WILL: Are you asking him do most Board-certified pathologists belong to the American College of Pathologists? Are you asking him does the American College give the Board-certification exam?
MR. BROWNSON: No.
Q. We have already established that you are a Board-certified pathologist in the States and you are not a member of the American College of Pathologists in the United States.
A. Yes.
Q. My question now is finishing that topic, would you agree that the American College of Pathologists is the principal organization of Board-certified pathologists in the United States?
MR. WILL: What do you mean by

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"organization of"? That's why I'm asking the question, do you mean that most Board-certified pathologists belong to that organization?
Q. Would you agree to that statement?
A. I don't know.
Q. You do know what the American College of Pathologists is?
A. Not exactly.
(Ilgren Exhibit 9, excerpt from CAP Directory, marked for identification, as of this date.)
Q. I show you now Exhibit 9, which is a photocopy of the cover and certain entries from the 1994 directory of the College of American

Pathologists. On the cover it reads "The mission of the College of American Pathologists, the principal organization of Board-certified pathologists, is to represent the interests of pathologists, patients and the public by fostering excellence in the practice of pathology worldwide."

Do you see that statement?

A. Yes.

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Q. Having read that, do you agree with the statement contained on the 1994 Directory of the College of American Pathologists that they are the principal organization of Board-certified pathologists?

MR. WILL: I object to the question. You're asking him to agree with something on the basis of having read a self-serving statement on this and that's entirely inappropriate. He has no foundation to agree or disagree with the self-serving statement of this group.

MR. BROWNSON: Regardless of the objection, I would like an answer.

THE WITNESS: Would you repeat the question.

(The record was read.)

MR. GERSON: I also object to ambiguity and vagueness. The problem is no one is saying what they mean by the word "principal" in this case, so I don't see how the witness has a basis for agreeing or disagreeing without further explication or definition.

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A. If that's what they say. You might argue with the College of American Pathologists.

Q. Let's go back to your CV. First of all, since you have been at Bryn Mawr, Pennsylvania, as I understand it, you have no laboratory facilities at the apartment in Bryn Mawr, correct?

A. Yes. A microscope.

Q. What type of microscope is that?

A. A Bausch & Lomb. Just a light microscope.

Q. Going back to the CV on page 1 where it says "Chronology of Research and Professional Experience," this is Exhibit 1, it indicates that you got your doctorate of medicine in 1971. Is that correct?

A. No, that's '74.

Q. That should be 1974?
A. Yes. That's a mistake.
Q. So that's a typographical error?
A. Yes.
Q. The entry just below that indicates visiting resident, the Memorial Hospital for Cancer and Allied Diseases, et cetera, 1973.

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How can you be a resident before you have received your M.D. degree?
A. Again that should be '74. I was working with Dr. Jones on a project to deal with choriocarcinoma and malignant trophoblasts.
Q. Would it be fair to say that that was after you received your M.D. degree in 1974?
A. Yes, that's right. That should be '74 as well.
Q. So the entry on the CV should read not 1973 but 1974?
A. Yes, it should.
Q. As I understand it, you did your residency following your medical school in New York City, is that correct?
A. Yes.
Q. That was at NYU Cornell hospital?
A. It's at Cornell University Medical Center/New York Hospital.
MR. GERSON: The New York Hospital is not the same thing as NYU.
Q. So this was Cornell University Medical Center at New York Hospital in New York City?
A. That's correct.

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Q. When did you complete that residency?
A. I think June '76.
Q. Is that indicated on the CV here somewhere?
A. Possibly.
Yes, it's on page 3 under "Employment." That was right. It runs from June 1974 to June 1976.
Q. That's the first entry under "Employment" on page 3?
A. Yes.
Q. Was that residency actually completed in June of '76 or did you leave to complete your residency elsewhere?
A. That was the anatomical pathology residency.
Q. So it actually was completed at Cornell University Hospital in New York City?
A. Yes.

MR. GERSON: Cornell University Medical Center in New York City.
Q. Do you recall at the time you were Board-certified in pathology whether it was a requirement that you have a three-year residency

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before you were eligible to take those exams?
A. 48 months.
Q. My question is, again going to the CV, where is the 48-month residency before Board-certification in 1977?
A. I have the correspondence with the Boards of Pathology, but I wrote to them my first year at Oxford, I think it was the end of 1976, because I hadn't taken the Board exam yet, and I asked them what would be the required number of months prior to sitting for the exam, and they said you need 48 months.
I asked them if, I guess it was about 18 months that I had worked at Rockefeller simultaneously with the Cornell University Medical Center, whether that could also be counted, and they said yes.
They said in addition they would accept I think the first six months of my training at Oxford, they would give an allowance I think up to six months for research.
They also counted the two months I think in medical school I had, a month or two of neuropathology, so it's sufficient, the American

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Board of Pathology reviewed this request and granted it.
Q. So is it fair to say what you did within a two-and-a-half to three-year period, you got credit for 48 months?
A. I would say three years, something like that.
Q. On the CV under "Employment" on page 3, the entry we were looking at where you did your residency in New York City, it indicates that you were assistant pathologist. Is that a title that you actually held while you were doing your residency?
A. I believe that's the title that's on the big certificate I got from New York Hospital. I don't think they called it intern. I think it was assistant pathologist.
Q. But would the practical word for that, if you will, be resident?
A. Yes.
Q. In the common parlance of resident,

that's what that assistant pathologist designation means?
A. I think if you wrote to the hospital

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and asked what position I held during the two years, they would probably say assistant pathologist.
Q. Then it says anatomic pathology, and I assume that that refers to not a position at the hospital but to the specialty you were studying?
A. Yes.
Q. Then it says registrar/senior registrar grade.
A. Again that's for the sake of the English, to try to give them an idea of what all those different names are.
Q. Is it fair to say that in England, what we call a resident in this country is referred to as a registrar?
A. Yes.
Q. Are you familiar with the classification in America of senior resident?
A. Yes.
Q. Did they have senior residents at Cornell University Medical Center at the time you were a resident there?
A. Yes, they did.
Q. Were you a senior resident?

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A. No.
Q. Is it your testimony that your status as a resident there was equivalent to a senior registrar grade in England?
A. They don't translate terribly well because the systems are quite different, the postgraduate systems are quite different. There could very well have been things I was doing in the second year at Cornell that certain senior registrars would be doing, so it's just difficult to translate from the American to the English system.
Q. Do you know if you were Board-certified in pathology in 1976 or 1977?
A. I think it's probably '77.
Q. It says '76 on the CV.
A. That should be changed. I'll change that.
Q. As Board certifications go, were you relatively young when you received that certification?
A. I think I was the youngest in the history of the United States.

Q. Do you know that for a fact?

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A. I think somebody told me that once.
Q. It also indicates on page 2 of the CV under the heading "Research And/Or Professional Chronology" that in 1976 you were a visiting worker in the Imperial Cancer Research Fund.
A. Yes.
Q. Would it be fair to say that at some point in 1976 you moved to England, is that how that worked?
A. I arrived Guy Faulkes night, November 5, 1976.
Q. Was that for a visit or did you actually move there?
A. I went to do my Ph.D. there.
Q. Upon arriving did you go directly to Oxford?
A. Yes.
Q. Upon arriving there at Oxford, was the first position you held that of the visiting worker in the Imperial Cancer Research Fund?
A. No.
Q. What does it mean when you say visiting worker in the Imperial Cancer Research Fund?

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A. It meant that I was going in the late winter and probably for a portion of 1977, I was going down to the Imperial Cancer Research Fund to study the tumor slides in the so-called TRC, the tumor reference collection, that Rupert Willis had placed in the ICRF or the cancer fund, and it wasn't as if I was doing active research, though in fact Dr. Pang. who actually took care of the collection, you can see at the end here, it's Professor R.A. Willis and Dr. Lillian Pang, she and I did subsequently write a paper together and we used some of the materials, I think there were some lung cancers of dogs and cats that we incorporated into a monograph that I wrote.
Q. Was that a paid position?
A. No, that's not a paid position.
Q. Did someone ask you to do that or how did you come to do that?
A. It's a funny story. After I wrote to Chicago and I got permission to go ahead and take the exam in American pathology, there was a major problem that I was working largely in a basic science lab in the zoology/botany environment, but I needed a very fine tumor collection review

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for the test, so I had a series of these textbooks by Rupert Willis and I opened the page up and I saw that he was at Leeds, so I sent a letter off to Rupert Willis and said because he was a renowned tumor pathologist, I thought he had a good tumor slide collection that I might review.

I wrote to him and he said sure, you can come and see me and talk to me and you can review my collection that's now under the supervision of Dr. Pang at the Imperial Cancer Fund.

So I called Dr. Pang up and she wasn't in charge of the pathology department per se, but they said sure, come on down and we'll help you go through the collection.

Q. Do you recall if as a requirement to get your complete 48 months to sit for the Board certification in pathology in the United States, that when you were trying to get credit for the months in England they said no, you have to do some specific pathology-related work and that's how you got on to this idea?

A. No, it was just, it was more of a

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study aid to get probably the finest tumor collection in the entire world just so that I could go through it, as most people do in preparation for exam, but no one said you need additional pathology experience. That was already taken care of.

Q. So just so I'm clear, when you wrote the Boards or when you wrote to the people who were giving the Boards for pathology in the United States and said I want to get credit for these months I spent over in England toward my 48 months, they didn't come back and say well, no, you're in a basic sciences laboratory and that's not really pathology, so you have to do some pathology work?

A. No, they liked it. I mean a lot of the work was fundamental biological work, but it was clearly applicable to pathology and tumor biology.

Q. Then it says in 1977 visiting scientist. University of Southern California. San Diego Zoo Society Hospital. Department of Pathology.

Did you actually move to San Diego in

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'77 or did you go there for a visit to compare

the tumor collection there with the one in England for purposes of that paper?
A. Again, that's another funny story. Briefly, it partly came about because I had to go to San Diego to take my Boards in pathology and I didn't have any money. So I went across the street to the Sir William Dunn School of Pathology because there was another American who was a third-year resident in pathology at the time on leave from Columbia, and I asked him do you know anybody in San Diego and he said well, there is this Professor Benirschke who is doing some very interesting work.
I wanted the name of someone who might sponsor a fellowship application, so I wrote to Benirschke, I was quite open with him, I said I need to come to San Diego to take my Board exams and I would like to apply for a fellowship. I found out that the UICC had these traveling fellowships and there was one called an ICRETT, they paid you for three weeks' living and paid your travel expenses, so I wrote a number of applications with Benirschke which gave us the

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funding to fly me over to San Diego.
It was quite a nice time. I went and took my exams for the first two days and then Benirschke and I sat down and worked up the details of my research project.
Q. Would it be fair to say that this entry, visiting scientist, was this project you did in San Diego?
A. Yes.
Q. To fund your trip over there to take the Boards?
A. That's what they called me, a visiting scientist.
Q. How long were you actually there visiting in that position?
A. I think three weeks.
Q. Did that then result in this publication of Ilgren, Griner, Benirschke and Pang, "A Comparative Study of Pulmonary Tumors"?
A. Yes, it did.
Q. Basically that was a study of tumors in animals at the San Diego Zoo compared with these ones over in England?
A. Comparatively speaking, it was

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actually animals versus humans, but it incorporated domestic species held at the Cancer Fund in London with the wild animal species that

were held in San Diego, so the comparison really refers to animals versus humans.
Q. Does that research form any basis for your opinions concerning asbestos and disease?
A. I would have to read the paper again. I haven't read it for a long time.
Q. I was afraid you would say that, because I read the paper at my office yesterday and forgot to bring it with me. I guess we don't have a copy here, is that correct?
A. I don't have one.
Q. So without reading the paper you couldn't answer that question as to whether it forms a basis of your current opinions on asbestos and disease?
A. Do you have a specific question you wanted to ask me about it?
Q. I'm just asking whether the research which is described in that paper forms any part of the basis of your current opinions concerning asbestos and disease.

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A. Again I would have to read the paper and see what I put in it.
(Recess taken.)
BY MR. BROWNSON:
Q. Are you currently affiliated with any hospitals?
A. No.
Q. Do you have staff privileges at any hospitals?
A. No.
Q. Are you currently engaged in the practice of medicine?
A. Reviewing pathology slides and doing postmortems?
Q. Right.
A. No. I mean I do review, in consultation, pathology slides that are sent to me often for litigation purposes.
Q. So if some lawyer representing a company in asbestos litigation would want you to review slides, he would send those to you and you would look at them?
A. Or for a benzene case or an arsenic case or whatever the case happens to be.

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Q. But as a layman I make a distinction between what I guess I would call a research person and a person practicing medicine seeing patients day to day, or in the case of a pathologist being in a hospital and seeing

pathology generated by the hospital?
A. Right.
Q. You are more in the research category.
A. In the Brownson sense, I am not.
Q. Would that be a fair characterization?
A. As you have described it, yes.
Q. Are you affiliated with any medical group of doctors?
MR. WILL: You mean like a clinic or pathology group?
Q. Are you affiliated with any clinical practice, clinical medical practice?
A. Like an HMO group or something?
Q. Right.
A. No.
Q. Are you on the staff of any colleges or univerities in any capacity in the United States?

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A. No.
Q. Since you came back from Oxford to the United States, have you been on the staff of any hospitals here in the United States?
A. No.
Q. Have you been on the staff or faculty of any colleges or univerities or medical schools in the United States?
A. No.
Q. Let me go back to the CV. We were on page 2 where we were under "Research And/Or Professional Experience Chronology."
We talked about this thing at San Diego. The next thing you list in 1977, traveling research fellow, International Union Against Cancer, Geneva, Switzerland. Do you see that?
A. That was effectively the fellowship that sent me to San Diego. That was my title from the fellowship, this ICRETT thing.
Q. So this was the group, the International Union Against Cancer?
A. Right.
Q. That sponsored this thing in San Diego

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that you just talked about?
A. The UICC, which is the UICC standard asbestos, that's a research body analagous in some sense to the WHO or whatever, and they fund research fellowships, so you just apply to them and they send you an application.
Q. So the traveling research fellow

listed there in 1977, would that be the same as the visiting scientist in 1977?
A. Yes.
Q. You were never actually stationed or living or working in Geneva, Switzerland, were you, in 1977?
A. No.
Q. Then the next entry is 1976/77, which is post-doctorate research fellow, et cetera, Department of Zoology.
That's something different, as I understand it, than the thing in San Diego?
A. Yes.
Q. When you moved to England after completing your residency in New York City, you arrived there in '76, and was this then what you started doing?

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A. I needed funding to pay for the tuition cost and also to simply live on. So when I first arrived there, I applied for a series of grants or fellowships, one of which was sponsored by the World Health, another was American Cancer, another was NIH, and most of them came through, so this was the first grant that I had applied for which was in the International Agency for Cancer Research Division of the World Health Organization, which is based in France in Lyon, and that was a one-year grant and I don't think I took the full year because then the American Cancer Society gave me a two-year grant, but I couldn't keep the WHO grant at the same time as the American Cancer Society grant, so I stopped -- I can't remember, it was about eight months of the WHO -- and then I started with the American Cancer Society.
Then after a certain number of months I was given a three-year NIH award, so I stopped the American Cancer Society. So they more or less, it wasn't as if WHO ran for a perfect year and then the American Cancer Society ran for a perfect two years and NIH ran for a perfect three

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years. That was the kind of thing.
Q. Let me see if I can summarize it.
You arrive in England in 1976, go to Oxford to enroll in the Ph.D. program. You're in the Ph.D. program, and I mean D.Phil., you're in the program and you get the Ph.D. in 1980, correct?
A. I think so.
Q. During the time you're in England at

Oxford getting the Ph.D., what we see here looks like five entries on your resume which are entries for research funds that you got from these various organizations, would that be fair to say?

A. Yes.

Q. Were these research funds actually paying you to do some work or were they paying your tuition and expenses? Were they like scholarships or actually like a job where you go to work and get a paycheck?

A. That's a funny question. As I recall, they were paid monthly and they were to cover my living expenses, my -- I used some of the funds to pay for the tuition. Some of the research

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expenses like go down to the store. In each department they have a departmental store where if you need a bottle of formaldehyde or whatever, you have to buy that, so as I recall, I think they paid the department monthly to cover certain research expenses and they would pay me, again it's a long time ago, but I think they would give me a paycheck or whatever it was to pay me just to cover rent or food or whatever it happened to be.

Q. I'm familiar with the term scholarship, if you're in college or graduate school you get a scholarship or fellowship.

Was this an equivalent of that, where you were getting in the layman's term scholarships or fellowships from these various organizations?

A. I don't know how to interpret it. Part of the problem is I had already finished so much work beforehand that I was already a certified specialist, as it were.

Q. In pathology.

A. Then I literally wanted to start from ground zero again to get this basic science

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experience in a Ph.D. program, so in one sense I was a Ph.D. student and in another sense I was a kind of something else physician, so if you want to call it a scholarship. I tried to put down what they called the things.

I applied for a traveling research fellowship. I applied for a post-doctoral research fellowship with WHO. I applied for these special post-doctoral research fellowships, American Cancer Society.

I applied for the research fellowship

at NIH. That's basically what they call these things that you got, so I assumed if I was given a research fellowship, I was the research fellow.
Q. So these things that you have listed between 1976 and 1980, are those things that you applied for or things that you actually received?
A. They were things I applied for and then received.
Q. So there weren't any of these entries that you applied for and you didn't actually receive a fellowship or grant between 1976 and 1980?
A. Just ask that again.

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MR. WILL: Are you asking did he receive all of the things that are entered on that?
Q. Did you receive a financial grant of some sort from each of the five things you list between 1976 and 1980 while you were getting your Ph.D.?
A. I received, I can't remember the exact money amounts, but I received several thousand dollars for the traveling research fellowship. The World Health Organization fellowship I think was $6,000. The special post-doctoral research fellowship was I think $10,000 or $11,000 a year for two years. The research fellowship for NIH was, I can't remember. I think it was $15,000, $16,000 and $17,000 a year.
Q. 1970 to 1979, it says research fellow, NIH.
A. Right.
Q. The NIH is the National Institutes of Health in the United States?
A. Yes.
Q. So basically what you got was, if I can use layman's terms, a scholarship or grant

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from the National Institutes of Health in the United States to help fund your Ph.D. education and research at Oxford?
A. Right, plus the post-Ph.D. work too.
Q. Look at the entry 1979-1980, visiting scientist, Sir William Dunn School of Pathology, University of Oxford.
First of all, did you hold the title of visiting scientist in the School of Pathology?
A. I think that was the title. When I finished my Ph.D. or D.Phil., actually during the six-month period before I had received the Ph.D., I guess he was the regius professor of pathology

at the time, Henry Harris, wrote me a letter and asked me to come and see him.

He said if you would like to spend a year of continuing your work at the Sir William Dunn School, which is right across the street from the zoology department, we would be happy to give you lab facilities and make available basic research things. That was it.

Q. Was this a paid position from the University of Oxford?

A. That was again using more of the NIH

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monies. I had only used say less than a year of the NIH research monies to pay for my last year of the Ph.D. or D.Phil., 1979 to '80, and then I had, I think it was two years when I finished my D.Phil., I had two years of monies remaining on the NIH grant.

Q. So you applied some of that money to this position?

A. The first year I split between the Sir William Dunn School of Pathology and the botany school, and then the last year of the NIH was the first year in neuropathology.

Q. That other CV that we had marked as Exhibit 3, if we can drag that out, I'm looking at the second page of it and I see an entry from 1979 to 1980, it says visiting research worker, NIH, Sir William Dunn School of Pathology.

Is that the same thing as what we just talked about?

A. I guess so. I suppose it's the same.

Q. So on one resume you referred to it as visiting research worker and then on the more recent one you referred to it as visiting scientist.

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A. Right. I probably abandoned two words for one for the sake of economy.

Q. Is visiting scientist or visiting research worker an actual title at Oxford or are those descriptions you have inserted into the CV to describe what you were doing?

A. They are descriptions to convey what I was doing, largely, at the time.

Q. You received a license to practice medicine in the State of New York in 1975, is that right?

A. Yes.

Q. Have you been licensed in any other state to practice medicine in the United States?

A. No.

Q. If you look at page 1 of your CV, numbered page 1 on Exhibit 1, the recent CV, it indicates you are licensed in medicine and surgery, State of New York in 1975, correct?
A. Yes.
Q. And then two entries above it says 1975, diplomate of the National Board of Medical Examiners.
Is that something different?

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A. When you pass your national boards you get a diploma and basically everybody who, as far as I'm aware, that gets a license has to pass the national boards, so that just meant I passed the national boards.
Q. Did you ever obtain a license to practice medicine in England?
A. I had or still have, I believe, the equivalent license, which if you look on page 2, 1982, General Medical Council full registration, so they say you're registered to practice there.
Q. So the entry 1982, General Medical Council full registration, in layman's terms means a license to practice medicine in England?
A. Yes.
Q. Is there some distinction between full registration or full license and some lesser registration?
A. You have to be fully registered. You have to be.
Q. Then if you look at the next entry, as long as we are in 1982, it says locum, consultant, neuropathologist, Radcliffe Infirmary, Oxford. Locum, as I understand it, is

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from the Latin, and basically it means holding the place of someone else?
A. The boss was away. He let me run the shop.
Q. That's indicated June 3-7, 1983?
A. Yes.
Q. So I'm trying to figure out what that was. Would it be fair to say that for a couple of weeks in June of '83 the boss was away at the Radcliffe Infirmary and you were able to fill in for him?
A. I suppose I ran most of the department in his absence. It was kind of exceptional circumstances that they would let one do that.
Q. Why would that be exceptional? Doesn't somebody have to be the neuropathologist when the boss is on vacation?

A. But it's an extremely responsible job. They had a catchment area of 3,000,000 people. It's one thing to be a training neuropathologist with world class, very senior people around with whom you can show a slide, but if there is no one around and you have to tell neurosurgeons sure, that's this, you have to

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treat them this way, I think as a training neuropathologist that's a pretty weighty responsibility.

Q. During that two-year period of time did you actually --

A. Two weeks.

Q. During that two-week period of time did you actually see neuropathology material from patients at the Radcliffe Infirmary?

A. Always. I didn't leave the infirmary. I was just there in the same department. It didn't mean I went to a peripheral town.

Q. Let me see if I can summarize your experience. After you obtained the Ph.D. in 1980, it looks like you then went to work in terms of your day-to-day activity and worked at the Radcliffe Infirmary, is that fair to say?

A. Yes.

Q. At the Radcliffe Infirmary you went to work as a pathologist and in particular a neuropathologist, correct?

A. Yes.

Q. Was that actually, to use the American

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equivalent, a paid doctor on the staff of that infirmary, is that what that is?

A. Yes.

Q. Is that like a salaried position?

A. Yes.

Q. They have the national medical, that's why I'm asking the question, it's not an independent, free-standing medical group or hospital of some sort.

A. National Health Service set salary scale, everything.

Q. The Ph.D. you obtained in 1980 and the thesis was what?

A. Title of the thesis?

Q. I'm asking for the topic of the thesis.

A. I have a description here. It's on page 10. Doctoral dissertation summary.

Q. Would the summary on page 10 be at

least what you're presenting at the present time on your CV as the summary of your doctoral thesis you obtained in 1980 in the Department of Biology and Zoology?
A. Zoology, yes, that's the summary.

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Q. Did the work leading up to that Ph.D. include any work with asbestos?
A. Did I do any asbestos work here?
Q. Right.
A. No.
Q. In the Ph.D. thesis itself, first of all is that published somewhere?
A. In I think twelve different papers. They are listed in the first portion of the bibliography.
MR. GERSON: In 12 different publications, did you mean?
THE WITNESS: Yes.
A. It would be papers Nos. 3, 4, 5, 6, 7, 8, 9, not 10, 14, 15. That's it. It looks like nine publications.
Q. So those nine publications that you have listed which are all on page 12 of your CV as Nos. 3, 4, 5, 6, 7, 8, 9. 14 and 15, were publications resulting from the Ph.D. dissertation?
A. Yes.
Q. I assume that you didn't, they don't each parrot the same dissertation nine times.

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Those are different publications resulting from that Ph.D. work?
A. All different datasets. 14 is a review article which reviews the whole field, but the rest have individual datasets. There is not a great deal of replication and overlap.
Q. So is the entire thesis actually published somewhere in the form of a thesis in the literature?
A. It's published as these papers.
MR. WILL: Is the thesis itself entirely published in one place in the form in which you submitted it?
MR. BROWNSON: Right.
A. It's in the university library.
Q. But my question is was it actually published in the peer review medical literature?
A. Sure, in these journals.
Q. The nine articles you have listed and just told us about are published papers in the scientific literature which were as a result of

your Ph.D. research?
A. If you look at the back of my thesis, it will say, again I would like to have it with

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me, but to paraphrase what I think it says, this thesis was compiled according to clause such and such of the University of Oxford regulations whereby an individual may bind publications.
The usual route is not to do that. It's an exceptional way, but basically I didn't have the time and money to spend an additional year sitting around writing up everything as papers, and my professor basically encouraged us to write up as we went so that if we did experiments from say September to December, whenever it was, in January the following year, just sit down and say now I'm going to write that up for publication. You wouldn't stop doing your experiments. So it just saved -- I mean most Ph.D. people do their thesis, put together a conventional thesis, send it in and then they go back and write it up.
Q. You didn't do that. What you did is you submitted nine papers that had been published in the literature as you have just described, bound them together and submitted those nine papers as the Ph.D. thesis, is that right?
A. Yes.

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Q. Then that material was accepted as a Ph.D. thesis and you received a Ph.D. from Oxford in 1980?
A. Exactly.
Q. If we went and blew the dust off some shelf in some library at Oxford and found the thesis, it would consist of those nine papers bound together?
A. It would actually be the galley. They are not the final papers, but they are sort of the galleys.
Q. Galley proofs of those papers?
A. Or the submitted manuscripts.
Q. Did you receive any English degree or accreditation as a neural pathologist?
A. Just the associate MRCPath.
Q. So I guess what I'm trying to get at, there is not an actual medical degree or graduate degree from a university or a college that you received in neuropathology in England?
A. No.
Q. So at whatever point, and I think we saw it was October of 1981 that you received the

associate membership in the Royal College of
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Pathologists, that would be the date you could officially call yourself a neuropathologist in England, would that be fair to say?
A. I imagine so.
Q. In terms of actually practicing neuropathology, would you need that associate membership in the Royal College of Pathologists to go to the Radcliffe Infirmary and be a neuropathologist?
A. I can't remember the exact requirements. I think it depends to some extent on the hospital. I can't remember the actual requirements within the English system, whether they would want you to have the full membership or whether you could have had, such as I did, a certain number of years of experience and have seen so many thousand brain biopsies, done so many neurological autopsies and had the American boards. I don't know how they would read that. I don't know what the standard rules would be.
Q. Just if we could try to summarize your medical accreditation or status as of October of 1981, would it be fair to say that as of that point you were a neuropathologist in England,
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whatever was required to do that?
A. Plus I was doing all the forensic pathology, most of the forensic pathology for the Radcliffe Infirmary as well, so I would say in inverted quotes a forensic pathologist and a neuropathologist.
Q. Have you ever received any certification or accreditation as a neuropathologist in the United States?
A. No.
Q. So going back to the original Board certification you got in pathology in 1977, that was just anatomical pathology?
A. Yes.
Q. Up until October of 1981 when you began your "practice" as a neuropathologist in England at the Radcliffe Infirmary, had you done any medical work with respect to asbestos?
A. Medical work with respect to asbestos means what?
Q. Up until that point had you seen any patients clinically who had asbestos disease?
A. As a medical student I believe I saw a few cases of mesothelioma when I was doing my
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residency at Cornell. I went through the James Ewing slide collection, which was 20,000 cases, and they had different examples of mesothelioma there, but I didn't work in a clinic where people were coming in for asbestos-related disease.
Q. As of that point in time had you ever reviewed any x-rays as part of a clinical practice of people with asbestos?
A. No.
Q. Up until that time had you reviewed pathology material, whether it's slides or actual pathology notes, of asbestos-related tumors as part of your work?
A. I had off and on. Again largely a few cases of asbestosis and a number of mesothelioma.
Q. That was during your residency and medical school training in this country?
A. It was either in medical school or residency training here or when I was studying or working, whatever you want to call it, at the cancer research fund in London. You're talking about before '81?
Q. Right. Would it be fair to say, however, that that was not a subject or a focus

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of any research you were doing before that time?
A. That's correct.
Q. If you would have seen such cases, it would have been, how would I say, I don't want to say happenstance, but as part of your broad experience in seeing many kinds of tumors and pathology materials in your training?
A. That's right.
Q. Have you ever done laboratory research in connection with asbestos? By that I mean something other than reviewing literature, meeting with people, looking at records, that sort of thing? Have you done research in the laboratory?
A. Yes, that's described on page 2, second entry from the bottom, and then I think I may have described that in more detail later on.
Very briefly on page 7. There is no publication arising out of that.
Q. Where is it described briefly on page 7?
A. Right in the dead middle there.
Q. This thing where it says Oxford University, 1985 to 1987, asbestos body chelated

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treatment project?

A. Yes.
Q. So to try to move this along, the actual laboratory research you have done concerning asbestos is shown in your CV in two places, one on page 2, second to last entry at the bottom, and then it appears again on page 7 in this entry in the middle from '85 to '87?
A. Are you asking me does this broadly apply to every -- let me say it differently. In terms of sitting in the lab and injecting rats, doing animal studies, specifically hands-on asbestos work?
Q. Right.
A. That's correct.
Q. I just want to ask some basic questions. The study of pathology is basically looking at tissue from the body, correct? That's what a pathologist does?
A. It's a compound question, really. What did you ask me, what was the first part?
Q. A pathologist, what a pathologist does is look at or study tissue samples, would that be fair to say?

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A. It could be tissue samples. It could be various fluids. It's a whole range of materials.
Q. Materials from the body?
A. A pathologist can also be an experimental pathologist. A pathologist can also be a research -- I think there is a whole array of categories of pathologists. The person who studies disease may be a fundamental research pathologist who never looks at a body, doesn't look at a whole organism, may spend his entire life doing in vitro studies or theoretical modeling.
Q. A pathologist does not treat living, breathing patients and put a stethoscope to their chest?
A. That's correct.
Q. The pathologist studies or analyzes samples of tissue and fluid and that sort of thing?
A. Pathology is the study of disease. A pathologist is a person who studies diseases.
Q. I'm not trying to make more of this than there is, but an epidemiologist also studies

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diseases, but a pathologist studies diseases from the perspective of looking at or studying or analyzing tissues and parts from the human body?

A. Sometimes.
Q. When you spoke earlier of breaking it down more specifically, some people are research pathologists who do research and some people are what would you call them, medical or practicing pathologists that actually look at the day-to-day tissue that comes down from the surgeon?
A. Hospital clinical pathologist, something like that.
Q. The specific specialty of neuropathology which you obtained in 1981 or thereabouts, how would that be described? How could you best characterize that specialty?
A. Neurological pathology is the study of diseases of the nervous system whereby one is responsible for the diagnosis of neurological diseases which might be inflammatory, tumorous, infectious, any agent or toxin, any agent that would affect the central or peripheral or autonomic nervous systems a pathologist might study if he is a neuropathologist.

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Q. Is mesothelioma, for example, considered a neurological disease that would be studied by a neuropathologist?
A. Not yet, I suppose.
Q. How about asbestosis, is that a neurological disease?
A. No.
Q. How about lung cancer, carcinoma of the lung?
A. No.
Q. Let's go back. I think before we get on this topic we were talking about the way, what you described as the hands-on research. That was this project beginning in 1984, I guess, with Professor Shubik?
A. Yes.
Q. It looks like it started in 1984 and continued through '87, is that right?
A. It was almost two years. It was about two years or so.
Q. At page 7 of the resume it indicates that it was from 1985 to 1987 and on page 2 it says the grant was received in 1984. Is that a correct chronology of things?

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A. We may have gotten the grant in '84 and the work began in '85. I can't quite remember. I know we worked on writing the grants and making application to the Health And Safety Executive and other agencies for the money. Then

I finished the senior registrar job July 1, 1985, so part of what I was doing from July 1, 1985 into '87 was working on this project.

Q. That was my next question. Senior registrar, that would be what we talked about before, equivalent to a resident in the United States?

A. In England you finish medical school. You do a year as a so-called house officer, which would be the old internship. Then you do one to two years as a senior house officer, SHO, and then you do three years as a so-called registrar, and then four years as a senior registrar.

That's the general progression, the two to three years of registrar. It can vary a bit. So I just throw that in so you have a better idea of the nomenclature and the way it progresses with house officer and SHO and registrar and senior registrar.

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The resident, most American training programs are shorter, but the medical school period per se is longer, where in England the medical school period is a bit shorter and the postgraduate training is longer, so there is a slightly more protracted, I guess it's just the way the society wants to emphasize either the pregraduate or postgraduate training.

Q. Could I characterize your work from the time you obtained the Ph.D. until you became a faculty member at Oxford in 1984, that interim of about four years, could I characterize that work as being a neuropathologist on the staff at the Radcliffe Infirmary with some forensic pathology on the side?

A. I also was given a grant from the Medical Research Council when I began at the Radcliffe Infirmary to do neuro-embryological research, so I was really doing three different things. Actually there were more, but three basic things. I was one, doing my neuro-embryological research, which was related to brain tumors. I was doing this forensic business and I was doing this sort of senior

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registrarship, diagnostic hospital/clinical neuropathologist.

Q. Would it be fair to say that your primary interest at that time was still trying to conduct this research but that you were kind of holding down two jobs to fund it, or were you actually practicing neuropathology or how would

you describe your status during that time?
A. I think there could easily be an analogy between an associate professor here who would have an NIH grant, who would be doing some research in his department and also doing some diagnostic work, either a pathologist looking at slides or as a clinician looking at patients, so it's just the main grant-giving body in England is the Medical Research Council. It's the equivalent of NIH.
Q. From 1980 to 1981 were you a faculty member of any college or university in England?
From 1980 to 1984, until 1984, were you a faculty member of any college or university in England?
A. The last page of my -- I thought I had a photocopy of the faculty membership

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certificate. I don't remember when that was dated.
Q. That's a good point. Actually if you look at CV No. 2, I think that's the one that has it. Let's look at that.
A. I think it's '84. Here it is.
Q. Where do you find it?
A. It's in the very back of Exhibit 3. It's dated 31 January 1984.
Q. So on January 31, 1984 you became a member of the faculty at Oxford University, right?
A. That's what it says.
Q. My question is before that date, from 1980 when you got the Ph.D. until January 31, 1984, were you on the faculty of any college or university in England?
A. No.
Q. Were you doing any teaching of medical students during that interim from 1980 to January 31, 1984?
A. I was giving the university lectures in brain trauma, brain infection, brain demyelination. I gave either four or five major

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lectures on brain diseases in the pathology course at the University Medical School. It was part of their curriculum.
In England when you finish high school, as it were, you go into medical school. It's three years of a preclinical medical training, then three years of a clinical medical training, but you're still in medical school.
In the first three years, I think it's

in the third year, they are required to take a basic pathology course, and neuropathology and other pathological subspecialties are covered, and the neuropath department always participates in that teaching program, so, for example, in the general pathology department the experts in renal pathology would give a couple lectures on kidney disease. The experts on whatever would give their didactic presentation.

Q. So if I could again try to summarize in layman's terms, from 1980 until January 31, 1984 you were not a faculty member on the paid, full-time faculty of the medical school at the University of Oxford, but what you did is you gave some lectures to third-year medical students

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in the specialty of neuropathology, would that be fair to say?

A. And house staff and clinical medical students. There was a whole series of different grades of students, of house staff.

Q. House staff at the Radcliffe Infirmary?

A. Yes. Other residents, we also had to make certain presentations to the entire department, so there is a whole different series of training or teaching.

Q. Does that appear on the CV, that particular experience?

A. Which experience?

Q. That you have just mentioned where you gave the lectures in neuropathology to the medical students or the house staff?

A. No.

Q. This reference in 1983 of subfaculty member, department of biochemistry, University of Oxford, that's something different?

A. I just took this off of what they sent me. That should be '84. I'm sorry it's so complicated. Look at the back of Exhibit 3.

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You're a member of a college. You're in that faculty, but you're a member of a subfaculty, because obviously that biological and agricultural sciences has a number of subfaculties, so in that they would have, they probably had physical chemistry, organic chemistry, biochemistry, zoology, cellular pathology.

So for each subfaculty they would have maybe 30 people. I can't remember exactly how many. Then you would have subfaculty meetings

every two or three weeks. They would talk about who is doing what research, how they should revise what aspects of the curricula, what should they be teaching, what shouldn't they, all sorts of things.

Q. On January 31, 1984 you became a member of the biological and agricultural sciences faculty at the University of Oxford and also at the same date you became a member of the subfaculty in biochemistry?

A. Exactly.

Q. So where it says that that occurred on your CV in 1983, that should be 1984?

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A. Yes.

Q. I would like to get back to the research project you did with Professor Shubik. That took place from 1985 to 1987, correct, and during that time period -- you were nodding your head.

A. I'm sorry.

Q. During that time period you remained a member of the faculty of the Department of Biological Sciences at Oxford?

A. Yes.

Q. What portion of your time during those years when you were on the faculty until 1987 was spent on that research project? Was that your day-to-day activity or was that half your time or how did that work?

A. It would be hard to estimate. Maybe 20 percent of my time, 25 percent of my time.

Q. As far as the grant that was awarded by the health and safety executive, first of all that's the health and safety executive in England?

A. Yes.

Q. As far as that grant, were you a

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recipient of the grant or was the recipient Professor Shubik, or was it both of you?

A. We were co-recipients on the grant.

Q. So if we went to the grant application and looked, it would list Ilgren and Shubik?

A. It should. I haven't looked at it, but it should list that.

Q. Would it list anyone else?

A. No, just the two of us.

Q. Do you know if Professor Shubik had received any previous grants from the Health And Safety Executive?

A. I don't believe so. I just don't

know. I don't believe so.
Q. Do you know who it was who came up with the idea for this particular research project?
A. I think it was largely Shubik in that it was a project based on the idea that there may be a two-stage initiation promotion type reaction that would occur between the iron and the asbestos fiber, whereby perhaps the iron as it bound to the fiber could potentiate its effects.
He and I obviously discussed the thing

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in great detail and modified the protocol, but I would think the fundamental ideas would certainly be his.
Q. How was it that you came to be involved in that project, since your prior experience had not been in this field?
A. I had a boss in neuropathology, Trevor Hughes, who was also bursar of Green College, and the head of Green College was Sir Richard Doll. The head of Green College, one of the other 38 colleges, was Sir Richard Doll. Sir Richard had invited Professor Shubik to be a proper fellow of Green College, a senior fellow, research fellow. He would have been a senior research fellow, an SRF, of Green College.
I was one of the rare Americans in Oxford, actually working in Oxford I should say. There are lots of Americans around, but actually working in Oxford, and Trevor Hughes must have told Shubik that there was an American working in his department who was terribly interested in cancer of all kinds, so Shubik invited me over to his house for a drink and we struck up a pretty good friendship and he knew I really enjoyed

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doing experimental work, and he was fundamentally an experimental pathologist.
So we were walking across, I remember we were walking across the park behind his house one day and he said well, how would you like to do an asbestos experiment? I said sure. That was how it started.
Q. Did Shubik receive his appointment on the faculty as a professor or don or whatever you call it at Green College?
A. Sure.
Q. I know he was affiliated with Green College, but what faculty department would that be?
A. He would be in the faculty of clinical

medicine.
Q. Is he still there?
A. I think so. I don't know. Pretty old now.
(Ilgren Exhibit 10, excerpt from World of Learning 1987, marked for identification, as of this date.)
Q. We have marked as Exhibit 10 a photocopy of a cover sheet and a section of a

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directory called The World of Learning, 37th edition, 1987.
What I did is I pulled out Oxford faculty. I'm trying to find out if we can find where Shubik --
A. No, this is too abbreviated. I shouldn't volunteer materials for you, but I could give you the full, they call it the Oxford calendar. It's a red book about 300 pages long. It lists all the proper fellows. This is a pretty bare-bones --
Q. Would it be fair to take it that what's listed here are the faculty members who have the rank of full professor, and it doesn't list the people who have lesser ranks?
A. No, there are other professors here for sure of all different kinds. I can send you the book.
Q. In any event, it's a bad list, an incomplete list?
A. Very abbreviated and incomplete.
Q. Would it be fair to say from 1985 through some subsequent period of years, maybe up to the present, maybe not. Professor Shubik was a

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professor in the department of clinical medicine?
A. No, you can't say that. He was a senior research fellow at Green College. To my knowledge, he would have had no professorial status whatsoever. He would have been on the faculty of clinical medicine and he would have had no teaching duties, no administrative responsibilities.
Q. So when we look at your CV, Exhibit 1, at page 2 and at page 7, where he is listed as Professor P. Shubik, we should drop the professor part?
A. He was a professor in Chicago at Chicago Medical School from 1953 to 1962, I think. The years might be slightly off. Then he became director of the Eppley Institute in Nebraska. I think he was an adjunct professor

with the University of Nebraska or whatever was there.

Then he became a visiting fellow at the University of Heidelberg, where I think he was a professor, but to say that he was an Oxford University professor at that time would be incorrect, so you could just call him Dr. Shubik.

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Q. So the reference on the CV to Professor P. Shubik at the time this study was underway should not be Professor, it should be Dr., because he wasn't actually a professor at Oxford at the time?

A. That's right.

Q. As I understand it, what this research project was was you and Dr. Shubik were trying to chelate the iron in the body to block the formation of asbestos bodies, would that be kind of a thumbnail sketch of what you were attempting?

A. And also thereby inhibit or at least ameliorate the pathogenic process. That's pretty much ahead of its day. The last couple of years with free radical ion, iron-related free radical ion theory coming out is a very important underlying mechanism in certain aspects of cancer, particularly in asbestos-related disease. As far as I know there was basically nothing in the literature that really suggested.

There have been subsequent papers, largely in vitro studies where they did manage, using chelators, to block a radical-related

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change. The problem here was a pretty simple one, that if you take a rat and you ask the question if I put asbestos fibers into that rat and I try to block by reducing its iron content so that there is insufficient iron going to the fiber, will the fiber still make disease.

In theory it's a great study but in practice you need to kill the rat, so to speak, because there is so much iron in the rat, it's almost impossible to deplete its stores whereby it wouldn't form.

Q. So again if I could try to summarize this, the intent of the project was to see if you could inhibit or prevent or ameliorate the formation of ferruginous bodies or iron asbestos bodies in the rat?

A. Right.

Q. By chelating the iron?

A. Right.

Q. What happened was in order to take enough iron out of the rat to do that, the rat died of iron deficiency?
A. The rats didn't die. The results were not clear. The rats didn't die. In other words

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there was a subacute phase and a chronic phase, but one couldn't --
Q. They got pretty loggy?
A. Nothing really untoward happened to the rats. It's just simply you could not say that there was less inflammation in the chelated-treated animals as opposed to the ones without.
There was one group of animals that for some reason seemed to get a kind of rebound deposition of iron, if I recall, with again as I recall a lot more inflammation.
Q. How many animals were involved?
A. I would have to get my lab notebooks. There would be hundreds over the time period.
Q. These were rats?
A. The rats, I can't remember if they were wistars or whatever.
Q. But this was not a human study, this was a study on rats?
A. This was a study on rats.
Q. I don't mean to make more of this than there is. I don't mean to stop you but I think I understand what the thinking was. I'm just

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trying to set some basic information about the study.
The animal at issue or used was rats.
A. Right.
Q. The asbestos was introduced to the rats by what, inhalation or injection or what?
A. This is how I met Chris Wagner, in fact. Shubik knew Wagner and we went to Chris Wagner's lab in Penarth to discuss the studies with him and also enlist his senior technologist, Rodney Hill, to help us do the intrapleural innoculation.
So we used, as I recall, I think chrysotile, amosite and then we also used erionite, which is not asbestos. I believe those three fibers types.
Q. Was that introduced into the rats by intrapleural injection, the different fibers?
A. Yes.
Q. Who actually did that work, was that you or Shubik or Wagner?

A. It was Hill, because he had such experience with the intrapleural injection technique.

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Q. In terms of the iron chelation, how was that physically accomplished?

A. We put an experimental iron chelator that we got from Procter & Gamble in the drinking water, a certain level, and we also periodically administered desferroxamine via tail vein injection, and there was a set regimen, drinking water, tail vein injection. Sometimes I would do tail vein injection.

Q. The object of that was to get the iron out of the rats, in simple terms?

A. Yes.

Q. Was the study funded in some part by Procter & Gamble?

A. As I recall, the whole study was funded by the Health And Safety Executive, which I think is the English equivalent of NIESH.

Q. Was there any participation in the funding by Procter & Gamble?

A. I don't think so. I can't remember.

Q. Was the study completed or what caused the study to end?

A. We just decided that for the reasons I listed, that it wasn't worth pursuing at that

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time.

Q. Was there any thought in starting this study or during the study that maybe this could be an actual treatment for people with asbestos bodies in their lung, it could be translated into a human medical treatment?

A. I think Shubik and I talked about that a lot in the context of trying to block asbestos body formation in people that might be acutely exposed in a building where all of a sudden in the newspaper in England you would occasionally read about children who were suddenly immersed in a cloud of asbestos because they walked into a contaminated room.

There was thinking about trying to make chelator therapy for acute exposure, something like this.

Q. But it didn't result in any human therapy for asbestos disease of any kind?

A. No.

Q. Did it result in any published literature in the medical or scientific literature?

A. No.

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Q. Were any reports issued to the funding agency, the Health And Safety Executive?
A. I think we sent at least one interim report in order to get the second phase of funding, but I would have to check.
Q. Was there a final report at the end of the study with conclusions in it?
A. I believe so.
RQ Q. If we wanted to get a copy of either the interim report or the final report, could that be obtained today?
A. You mean this second?
Q. Not this second. Does it exist somewhere today?
A. I suppose so. David Gompertz at the Health And Safety Executive was the liaison officer there, and as far as I recall we dealt with him. Shubik also dealt with at the time the head of the HSE, but the person really dealing with the grant mainly was Gompertz and he would be the contact person.
Q. You don't have a copy in your possession?
A. I might have it in my file.

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Q. You might or might not, you don't know?
A. Do you want a copy?
Q. I would like to get a copy.
A. I don't know. I'll have to --
MR. GERSON: We'll review everything Bob wants.
A. I will have to check and see if it's there. It's a long time.
Q. While you're making a note there, the fiber type that was injected intrapleurally into these rats was what?
A. I think the three types of fiber, chrysotile, amosite and erionite, but I can't remember if we also had crocidolite. I don't remember exactly. I think it was chrysotile.
Q. Do you remember the type of chrysotile?
A. I think it was UICC.
Q. Do you recall if any of these rats succumbed to tumors?
MR. WILL: Why don't you refer to your CV where it says secondary to crocidolite, zeolite and erionite.

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Q. We are referring to page 7?
MR. WILL: Yes.
Q. Mr. Will has pointed out that on page 7 it says crocidolite. zeolite and erionite, so does that indicate that the type of asbestos fiber was crocidolite?
A. It may have been. It's been a long time since I have done this stuff.
Q. Do you recall if any of the rats succumbed to mesothelioma tumors?
A. No. It was. I think they were six-week and twelve-week study periods, so it's not sufficient time for tumor formation.
Q. Do you recall the dose that was injected, the dose of fiber injection?
A. I think these were standard 20 mg inocula.
Q. So would it be a single 20-milligram dose in each rat?
A. I believe so. I would have to check. I think that's correct.
Q. With respect to Professor Shubik, are you saying that this experiment was the first work on the two-stage cancer mechanism or had

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work been done on that before this particular project?
A. In terms of asbestos-related disease?
Q. Let's start more generally. First of all, as I understand, Professor Shubik was a chemical carcinogen researcher, would that be fair to say?
A. Shubik is acknowledged as a co-founder of two-stage theory with Isaac Berenblum back in 1947.
Q. Before this project, wasn't Dr. Shubik concerned with chemical carcinogens as opposed to particles?
A. I think he did particulate work with Saffiotti in the co-administration of iron oxide particles with benzopyrene in hamsters to develop a lung cancer model. I know he had done some particulate research before that. but I think this was probably the first experimental investigation to test the hypothesis that iron may have a role in the pathogenesis of asbestos-related disease.
Q. So as far as Dr. Shubik's previous work on the two-stage mechanism of cancer. he had

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done some work with chemical carcinogens like

pyrobenzene and also trace metals? When you say particles --

A. I think he has written almost 300 papers, so he has gone through a fair gamut of chemical and physical carcinogens.

Q. Are you saying that Shubik hypothesized the two-stage cancer mechanism contemporaneously with Berenblum?

A. No, I think Isaac Berenblum was a reader in the Sir William Dunn School of Pathology and he came in in about 1927. One of his particular interests was investigating the factors that would serve as modifiers of tumor formation, so some of his early studies would be applying a carcinogenic. a carcinogen to a mouse and then burning the mouse. One study was carbon dioxide snow. Another was scratching the skin.

So Berenblum over the years before he met Shubik had developed the thinking about interactions between chemical carcinogens and irritants. Shubik came along and participated with Berenblum in the studies and was second author, co-author, so I would say that Berenblum

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probably laid down a lot of the theoretical model on which the study was done.

Q. In terms of chronology, would it be fair to say that Berenblum had hypothesized the two-stage mechanism before he began his work with Shubik? That's all I'm trying to get at.

A. That's right.

Q. So Shubik was not a co-founder contemporaneously in time of the theory?

A. Some people may shoot me for saying that, but I mean a lot of people give. I would think, Berenblum and Shubik equal credit for this, but my understanding would be that Berenblum added this additional material.

Q. You had mentioned that Shubik earlier had done earlier work at the Chicago medical school from 1953 to 1962. and in 1962 he left to go to the Eppley Institute in Omaha, Nebraska, correct?

A. Right.

Q. That's called something like the Eppley Institute of Medical Research? I don't know its exact name. but it's in Omaha.

A. Something like that. yes.

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Q. Are you aware of the fact that while he was at the Eppley Institute he had one or more grants from the National Cancer Institute?

A. I think he ran an NCI budget of $6,000,000 to $10,000,000 a year.
Q. Did he ever indicate to you that he had gotten in trouble with NCI auditors for commingling that grant money?
A. What a mess.
Q. You are familiar with that?
A. The big scandal, sure.
Q. Do you know if he ever was indicted for that?
A. I had the impression, I didn't know that when I first started working with him, I had the impression that there was some very sloppy bookkeeping at the Eppley.
MR. WILL: The question was do you know whether he was indicted. Either you do or you don't.
A. I do not know.
Q. Do you know if it was as a result of that scandal which may or may not have resulted in indictment that he left Eppley and went to

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Heidelberg?
A. I'm not sure, but it could very well have been so, yes.
Q. Once Shubik got to Oxford after, first of all do you know why he left Heidelberg?
A. I don't know why he left Heidelberg.
Q. In any event he came on the scene at Oxford as you have described. Once he got there he was doing other things other than this particular research project, wasn't he?
A. He was, I don't know if he founded the journal, I think it's Cancer Letters there, so he had one of his journal offices there. He had founded an organization called Toxicology Forum, and he ran it from his office there.
I think he estimated he spent 200 days a year in an airplane somewhere traveling to meetings and whatnot, so for the time he was in Oxford he devoted some time to our research project, some time to the journal, some time to Toxic Forum matters, some time to other matters, consulting.
Q. Do you know if Toxicology Forum published a newsletter while he was at Oxford?

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MR. GERSON: I object to the relevance.
MR. WILL: Why are we going through all this about Dr. Shubik?
A. I don't know.

Q. Did you ever appear as a co-author on any of the Toxicology Journal or toxicology newsletters that he published while at Oxford?
A. The newsletters? I don't know if there are newsletters.
Q. Whether there are or not, my question is did you author any articles in any of his publications that he edited while he was at Oxford?
A. I published one paper in Cancer Letters. I don't think he was, he wasn't a reviewer for that.
Q. Is that paper listed on your CV Exhibit 1?
(Continued on the following page.)

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A. Yes, that's a choriocarcinoma in vitro paper.
Q. Look at the publications beginning on page 12 and tell me where that shows up.
A. 25.
MR. BROWNSON: Why don't we stop there.
(Time noted: 5:50 p.m.)

_____
EDWARD B. ILGREN

Subscribed and sworn to before me this ____ day of _____, 1994.

_____

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CERTIFICATE

STATE OF NEW YORK)
: ss.
COUNTY OF NEW YORK)

I, DOUGLAS M. BURKE, a Shorthand Reporter and Notary Public within and for the State of

New York, do hereby certify:
That EDWARD B. ILGREN, the witness whose deposition is hereinbefore set forth, was duly sworn by me and that such deposition is a true record of the testimony given by the witness.
I further certify that I am not related to any of the parties to this action by blood or marriage, and that I am in no way interested in the outcome of this matter.
IN WITNESS WHEREOF, I have hereunto set my hand this ____ day of _____, 1994.

_____

DOUGLAS M. BURKE

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---------- I N D E X ----------

---------- INFORMATION REQUESTS ----------

DIRECTIONS:
RULINGS:
TO BE FURNISHED:
REQUESTS: 23, 30, 32, 204
MOTIONS:

---------- EXHIBITS ----------

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[redacted]

EXHIBIT

PIFF 15
11-15-91

Comments on the paper "Cytotoxicity of a Short-Fibre Chrysotile for Human Alveolar Macrophages; Preliminary Observations (1983). Yeager H, Russo D A, Yanez M, Gerardi D, Nolan R P, Kagan E, Langer A.M. Environmental Research; 30, 224-232.".

The paper presents a series of results which demonstrate higher cytotoxicity for a "short-fibre" chrysotile from Calidria than for the reference UICC chrysotile A from Zimbabwe despite a virtually identical number of fibres longer than 5 μm in the applied doses. Progressively milled derivatives of the Calidria fibre were even more cytotoxic even though there were no long fibres in the milled forms. This conclusion was presented as having important biological implications for the use of short fibre chrysotile in industry.

The Calidria fibre and the milled forms used by Yeager were those produced and described by Langer et al (1978). The UICC chrysotile was that prepared by Timbrell, Rendall and others in the late 1960s and described in the report of the Johannesburg Pneumoconiosis Conference (1970).

Langer et al. (1978) reported decreasing hemolytic activity for the Calidria fiber and the progressively milled derivatives of it. This result was unexpected because milling results in smaller particles, increased surface area, and increased particle numbers per unit mass, all of which are normally associated with increased biological activity including hemolysis. Langer explained this apparent contradiction by the observation that the milling had significantly changed the surface characteristics of the fibers and produced an impressive amount of physico-chemical data to support this argument.

The fiber lengths were measured by Transmission Electron Microscopy from high magnification photographs. The sample preparation for the TEM measurement involved dispersion of 1 mg of the bulk fiber in 0.1 ml of nitrocellulose (1% in amyl acetate). This preparation technique ought not to introduce any size selection in the sample, and all size ranges should be fully represented. Approximately 1000 particles were measured for each fiber sample with a specific set of fiber and fibril definitions viz: "a particle composed of at least two fibrils, the shorter being contiguous with and comprising at least one-half of the first, was called a fiber; objects counted as fibers possessed a length/width aspect ratio of at least 3:1; objects with smaller aspect ratios, composed of many fibrils, were counted as clumps; single fibrils, of any aspect ratio, including contiguous fibrils that did not meet the fiber criterion, were called fibrils.". These fiber definitions are not standard but would help to reduce inter-observer differences within that study. The minimum fiber length

recorded was 0.1 µm but no fiber diameter measurements were reported. A separate record was kept of the fiber or fibril nature of each particle and the data were presented in separate tables for fibers and fibrils each classified by length.

Yeager combined the data from the two tables of Langer et al. and simplified the length classes but did not explain fully how the mass/number indices were obtained. The numbers of fibers per microgram of dose were calculated assuming an average fibril diameter of 0.0333 µm and an average fiber diameter of 9 times this figure, i.e. 0.3 µm (Yeager did not explain that Langer et al had made this distinction). A particle with a diameter of 0.3 µm would have to have a length of at least 0.9 µm in order to be classed as a fiber but Yeager did not explain how the mass of fibers shorter than 0.9 µm were calculated even though Langer et al recorded more than 65% of fibers in the range of 0.1 µm to 1 µm for the unmilled Calidria fiber. The assumptions made about the diameters to be used for these particles would have an influence on the calculated fiber numbers per milligram.

Yeager et al. did not explain how the mass/number indices were calculated for the UICC chrysotile or what assumptions were made about the diameter distributions. They stressed the point about the absolute numbers of fibers in different length ranges in the actual doses used, correctly pointing out that 30% of 100,000 fibers is the same as 3% of 1,000,000 fibers. If this were genuinely the case for the fibers in this study then it might be very important. However there are good grounds for suggesting that the problem was oversimplified and too many assumptions were madein the arguments. For example, it appears that Yeager assumed that the diameter distributions of the UICC and the Calidria fibers were the same. There are no grounds for this assumption and since the diameter is a controlling factor in the calculation of numbers of fibers per unit mass it could make a critical difference.

Yeager used the length distribution data from the paper by Timbrell (1970) as the source of fiber numbers of the UICC chrysotile A. However, in contrast to Langer, Timbrell carried out TEM analysis of samples collected using a thermal precipitator dust sampling instrument from airborne dust clouds generated by a dust dispenser in a chamber. Timbrell did not describe his sample preparation methods but this dust generation and sampling method could not avoid some size selectivity for the sample as measured by TEM. Also Timbrell measured only particles greater than 0.2 µm, he provided no other indication of fiber counting criteria and made no distinction between fibers and fibrils.

Table 1 shows how different assumptions about fiber diameters can produce radically different mass/number indices for UICC chrysotile and Calidria chrysotile from Yeagers own length distributions. Fibers per unit mass for all fibers range from 1.4 million to 110 million for the UICC chrysotile and from 9.2 million to 540 million for the unmilled Calidria fibre. For fibers longer than 5 µm the numbers range from about 350,000 to 28 million. So if fiber numbers from Yeagers data were calculated using different assumptions about fiber diameter, then different conclusions might be made about the absolute fiber numbers although because the assumptions are made equally for both fiber types the relative fiber numbers are the same. The Calidria chrysotile would still have the same number of long fibers as the UICC chrysotile A, and still have more fibers of all lengths. However, since there were no actual diameter distribution data, it is difficult to see how fiber number comparisons can be made, given the ranges shown above.

Yeager et al. also cited a paper by Rendall (1970) with respect to the UICC asbestos minerals but they did not use the fiber length data of Rendall even though it was critically different from that of Timbrell (1970) and similar to the data for the untreated Calidria fiber of Langer (1978). As with Timbrell the lower length limit for measurement of fibers was 0.2 µm.

The data from Gibbs and Hwang 1980, which are more amenable to fiber mass calculations, show how strong are the influences of the dimensions of a small proportion of the long, thick fibers measured and that the actual fiber dimensions measured are critical to the calculated numbers of fibers per unit mass. In a pair of airborne chrysotile samples from mining and bagging operations they found that fibers longer than 5 µm constituted only 1.3% and 3.94% of the fiber numbers but 39% and 59% of the mass of the fibers. Any assumptions about fiber dimensions, diameters or lengths, of just a small number of long fibers in the sample have a dominant effect on the fiber numbers calculated The total fiber numbers for these samples of 1.8×10^7 and 1.2×10^7 per milligram respectively are controlled mostly by the diameters and numbers of these few long fibers. The statement by Yeager that the numbers of long fibers in the doses of unmilled Calidria fiber and in the UICC chrysotile are the same despite the differences in the total fiber numbers is then really only a function of the assumptions made about fiber diameters used in the calculations. The lesser importance of the short fibers in this calculation and an indication that the apparent difference between the two fibers may be largely a result of the assumptions made by Yeager are shown by the following calculations. If 25% of fibers shorter than 1.0 µm are added to the data of either Timbrell or Rendall and the fiber numbers per unit mass are recalculated then it shows that while the absolute fiber numbers increase dramatically (as expected) the numbers of long fibers per unit mass remain virtually the same. The numbers of long fibers per unit mass then are very insensitive to the fiber counting rules for short fibers or to the analytical methods that might influence the numbers of the finest and shortest fibers.

These two data sets produced by different observers (Timbrell and Rendall, both intimately involved in the production of the UICC asbestos) still differed by a factor of 2 despite apparently using the same methods on the same samples, indicating that conclusions based upon differing fiber numbers should be treated with caution.

Not only were the measurements of fiber dimensions by Langer er al. and by Timbrell and Rendall based upon different sample types and used different preparation procedures but they also used different criteria for inclusion of fibers in the count. In addition to this, the same assumptions about fiber sizes were wrongly applied to fiber populations with different length classes in calculating fiber numbers in the doses. There was in fact no sound basis for making comparisons of the size distributions or the fiber numbers of the UICC and the Calidria fibers and no basis for discussions about the relevance of fiber length to toxicity. The fact that large numbers (5×10^9) of fine Calidria chrysotile fibers were cytotoxic to human macrophages is scarcely surprising and the fact then that even larger numbers (1.15×10^{11}) were more toxic is even less surprising.

References:

Gibbs, GW, Hwang,CY. (1980) Dimensions of Airborne Asbestos Fibres. In Biological Effects of Mineral Fibres, ed. J.C. Wagner, Vol.1, International Agency for Research on Cancer, Scientific Publications No. 30, Lyon, 69-78.

Langer AM, Wolff MS, Rohl AN, Selikoff IJ. (1978). Variation of properties of chrysotile asbestos after milling. J. Tox. Env. Health, 4: 173-188.

Rendall REG. (1970) The data sheets on the chemical and physical properties of the UICC standard reference samples. In Proceedings of the International Conference on Pneumoconiosis, Johannesburg, ed. H.A. Shapiro, 23-27. New York: Oxford

Timbrell V (1970) . Characteristics of the International Union Against Cancer Standard Reference Samples of Asbestos. In Proceedings of the International Conference on Pneumoconiosis, Johannesburg, ed. H.A. Shapiro, 28-36. New York: Oxford

John Addison
Addison-Lynch
Edinburgh
25.1.94

Table2. Fiber numbers per microgram for UICC chrysotile calculated from the data of Timbrell and of Rendall (1970) and adjusted by the addition of 25% of fibers shorter than 1 µm.

UICC	Original Data	Short fibers added	Original Data	Short fibers added
Timbrell	3.6×10^6	4.9×10^6	0.9289×10^6	0.9287×10^6
Rendall	7.1×10^6	9.5×10^6	0.4333×10^6	0.4332×10^6

Table 3. Fiber number per microgram of chrysotile as used by Yeager, as recalculated for this review, and compared to other chrysotiles.

	ALL FIBERS	FIBERS L >5µm
YEAGER UICC A	0.1×10^8	2.5×10^6
TIMBRELL (1970) UICC A (Recalculated JA)	3.7×10^6	9.3×10^5
RENDALL (1970) UICC A (Recalculated JA)	7.1×10^6	4.3×10^5
YEAGER Calidria, unmilled.	1.1×10^8	2.1×10^6
LANGER Calidria Unmilled (Recalculated JA)	2.2×10^7	4.5×10^5
GIBBS & HWANG 1980 Mining	1.8×10^7	2.3×10^5
GIBBS & HWANG 1980 Bagging	1.2×10^7	4.5×10^5

Dr. E. B. Ilgren, MA, MD, DPhil, MACPath, MRCPath
Faculty of Biological Sciences,
Subfaculty of Biochemistry,
University of Oxford,
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Oxford

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Dr Charles Lorberau, PhD
Division of Physical Science &
Engineering,
Measuements Research Branch,
National Institute of Occupational Hygiene,
Cinn., Ohio

tel 513 841 4301 {fax 4500}

5 January 1994

Dear Dr Lorberau,

Thank you for offering to check your files in an attempt to identify the individuals to whom you may have sent the Calidria chrysotile samples. As discussed, I would be very interested to know of further data pertaining to fibre size, elemental analyses, and tremolite content. Thank you.

yours most sincerely,

EXHIBIT
PII-X16
11-17-94 M.

PARTIAL LIST OF PERSONS RECEIVING SAMPLE OF CHRYSOTILE (CH-29)
SOURCE: UNION CARBIDE, CALIDRIA ASBESTOS

copy of list received from Loberau of individuals who requested Calidria samples for research - original not found; date not known ca Feb 94.

Paul Laraia
Wastex Corp.
510 Heron Dr., Suite 107
Bridgeport, NJ 08014

David Rodriguez,
American Analytical Laboratories
100 Lincoln
Akron, OH 44308

C. Edward Howard
Center for Environmental Measurements
Research Triangle Park
Research Triangle Park, NC 27709

John FoyHoppe
Southern Illinois University
Dept. of Chemistry
Carbondale, IL 62901

Dr. Briant Davis
South Dakota School of Mines
Inst. of Atmospheric Sciences
501 E. St. Joseph
Rapid City, SD 57701

Alan Johanns
Ames Environmental
3910 Lincoln Way
Ames, Iowa 50010

EXHIBIT
PLFF K 17
11-17-94

Dr. John Roulston
DCM Science Labs
12975 W. 24th Pl.
Golden, CO 80401

Jerry Krause
Colorado School of Mines Research Institute
PO Box 12

PARTIAL LIST OF PERSONS RECEIVING SAMPLE OF CHRYSOTILE (CH-29)
SOURCE: UNION CARBIDE, CALIDRIA ASBESTOS

Golden, CO 80401

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Tech. Reseach Center
3422 South 700 W
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T Sgt. Mike Wantland
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Analytical Department,
Brooks AFB, TX 78235

Gregory Bromley,
Core Laboratories, Inc.
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Houston, TX 77040

Mr. William E. Gardner
Technology of Materials
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Santa Barbara, CA 93103

Dr. Lily Prigge
Microanalytical Services, Inc.
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Pasadena, CA 91101

Barbara Przybylska
EMS Laboratories
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South Pasadena, CA 91030

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Boars Hill,
Oxford

Dr. Claudio Martinelli

PARTIAL LIST OF PERSONS RECEIVING SAMPLE OF CHRYSOTILE (CH-29) SOURCE: UNION CARBIDE, CALIDRIA ASBESTOS

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37122 Verona, ITALY

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Fisica Dell'Ambiente
Via Salvo D'Agusto
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Dr. Giuseppe Scanarello
Istituto Di Medicina del Lavoro
Universita' degli Studi de Diena
Via dei Tufi 1
53100 Siena, ITALY

Dr. Luigi Paoletti
Laboratorio di Ultrastructure
Istituto Superiore di Sanita
Viale Regina Elena 299
00161 Roma, ITALY

Dr. Dinesh J. Parikh
Head, Occupational Hyg. Division and Asst. Director
National Institute of Occupational Health
Meghani Nagar, Ahmedabad, 380010
INDIA

FC M Hamilla 10/25/93

FIBER SIZE DETERMINATION

AND DISTRIBUTION OF

CALIDRIA CHRYSOTILE ASBESTOS

BY

TRANSMISSION ELECTRON MICROSCOPY

for:

Client: KCAC Inc.
P.O. Box K
King City, California 93930

Re: Fiber Size Determination and Distribution

Job Number: 8032

Date: November 30, 1991

Analysts: D. R. Miller
S. P. Breloff
B. E. Lisk
G. D. Browning

EXHIBIT
PIFX 18-94
11-18-94

Director: T. J. Spengler

ASTECO, INC.
PO BOX 179
MIDDLEPORT, NEW YORK 14105
(716) 735-3894

OBJECTIVE:

To determine the fiber size and distribution of Chrysotile fibers in 4 different samples of Calidria Chrysotile Asbestos using Transmission Electron Microscopy. The four samples being SG-130 (Standard Grade), RG-144 (High Purity Open), RG-244 and Short Fiber Chrysotile Ore.

PROCEDURE:

The samples were prepared for analysis by water filtration while trying to disturb them as little as possible and thus attempting keep any change in the fiber size of the sample to a minimum. A known amount of each sample was added to one liter of water and mildly shaken by hand to disperse the sample into suspension. A series of dilutions of each suspension was then filtered through 0.1μm pore size mixed cellulose esther filters and prepared for TEM analysis using a modified Jaffe wash and acetone condensation wash. The different dilutions of each sample were then evaluated for in the TEM for fiber loading and preparation acceptability, and one preparation was chosen for analysis.

Analysis begun on the selected preparation and proceeded until 400 fibrils were counted. Asbestos fibers which normally would be counted as complex structures (bundles, clusters or matrices), were examined closely and the individual fibrils which composed the structure were counted and measured individually as accurately as possible. The length and widths of the fibrils counted were measured at 20,000 and 60,000 magnification respectively. For each sample a morphology photomicrograph was taken of a typical fibril in each of three categories: $\leq$ 5μm, > 5μm but < 10μm and $\geq$ 10μm. It was assumed that all fibers in the samples were Chrysotile, therefore only one diffraction photomicrograph and EDXA spectra were obtained from each sample during the course of the analysis (the morphology of the fibers counted support this assumption).

After the analysis of samples SG-130, RG-144 and RG-244 were completed it was requested that an additional sample be analyzed. This sample was a portion of RG-244 which had not been treated with silicon by the manufacturer. Also it was requested that the original sample of RG-244, which had been treated with silicon be reanalyzed after steps were taken to disperse the sample better. The original sample of RG-244 was immersed in an ultrasonic bath for 30 minutes along with the new sample of RG-244. Both samples were refiltered and reanalyzed.

The short fiber chrysotile ore was analyzed using both PLM and TEM analysis. The course portion of the sample was first separated using a stereo microscope and forceps. The largest portion of this subsample was measured using the stereo microscope and ruler until it was no longer feasible. At this point the portions of the sample were immersed in refractive index liquid and analyzed using Polarized Light Microscopy until 400 particles were counted and measured. The remaining finer portion of the sample was then separated into two similar samples. One of which was suspended in water and filtered while the other was mortared and immersed in an ultrasonic bath for 30 minutes before filtration. Both samples were then analyzed.

CALCULATIONS AND DEFINITIONS:

Fibers were distributed into different categories according to their length, width and aspect ratio. For the purposes of this report a fiber is defined as a particle possessing parallel sides and an aspect ratio of 3:1 or greater where aspect ratio is defined as the numerical proportion of length to width with the width being defined as 1:

$$Aspect\ Ratio = x{:}1$$

$$x = \frac{L}{W}$$

where x - the aspect ratio

L - length of the fiber in microns (μm)

W - width of the fiber (μm).

Note: The larger the aspect ratio the longer the fiber.

Thus each sample has three distributions, one each for their length, width and aspect ratio. The mean, mode and median of each of the sample's distributions was also calculated. The mean used in this report is the arithmetic mean and is defined as:

$$\mu = \frac{\sum_{i=1}^{n} X_i}{n}$$

where μ - the arithmetic mean (length, width or aspect ratio)

n - the number of fibers counted

Xi- the quantity of the i^{th} fiber (length, width or aspect ratio)

i - the i^{th} fiber.

The median is defined as the middle variate for a population of variates arranged in either increasing or decreasing order for a population with an odd number of variates and the arithmetic mean of the two middle variates if the population has an even number of variates. The mode is the most frequently appearing variate in a population. In the report the mode is reported as an interval category of either length, width or aspect ratio.

Positive Skewness is defined as a distribution in which the majority of the population is smaller than the mean, and the largest component is below the midpoint.

REMARKS:

This report has been prepared for KCAC, Inc., and is not intended to be relied upon by others. Any reproduction of the report is to be in its entirety. The samples have been submitted to ASTECO for analysis and no sample collection activity has been performed.

All aspect ratios are referred to by the length or x component, thus a graph category of 3 - 6 would correspond to a range of aspect ratios from 3:1 to 6:1.

All length bar graph x values correspond to the midpoint of a length range. An x value of 1.75 would refer to a length range of 1.5 μm to 2.0 μm.

An assortment of graphs are included to help illustrate sample distributions and various samples comparisons. Graphs which refer to the short fiber ore sample as untreated are the of the sample which was ultrasonicated but not mortared, graphs which refer to the short fiber ore sample as ultrasonicated are of the sample which was ultrasonicated and mortared.

QUALITY ASSURANCE:

ASTECO is accredited by the American Industrial Hygiene Association for the analysis of asbestos. Our accreditation number is 356. This accreditation may be verified by contacting the American Industrial Hygiene Association at (216) 762-7294. The laboratory is also accredited by the New York State Environmental Laboratory Approval Program (ELAP) for the analysis of fibers and asbestos. Our accreditation number is 10834. This accreditation may be verified by contacting ELAP at 518 474-4471. The laboratory has accreditation by NIST (National Institute of Standards and Testing) for the NVLAP program (National Voluntary Laboratory Accreditation Program). This accreditation may be verified by contacting NIST at (301) 975-4016.

LIMITS AND CONDITIONS

1. In compiling this report ASTECO INC. (hereinafter We/Our) has followed procedures which we believe to be accurate and reliable but there may be other methods for evaluating asbestiform minerals which may produce different quantitative results.

2. The analytical results reported herein are not to be viewed as exactly equal to the results which may be found at different sampling locations due to the nonuniformity in concentration of these types of substances.

3. Any use of, or reliance upon, the data, or other information, contained herein by yourself, or any other party, shall be without any recourse to, or liability on Our part.

4. The breathing of asbestos dust may cause serious bodily harm and We can make no warranty or guarantee that the results contained herein indicate that safe levels of exposure exist. It is the responsibility of any user of asbestos or an asbestos-containing product to ensure that safe handling procedures are employed and that applicable government regulations are complied with.

DISCUSSION:

In the following discussion RG-244 SS will refer to the preparation of the original silicon treated RG-244 sample after it had been ultrasonicated, RG-244 SN will refer to the original silicon treated RG-244 sample which was not ultrasonicated and RG-244 NS will refer to the second RG-244 sample sent which was not treated with silicon and was ultrasonicated.

All three samples of the milled chrysotile which was originally submitted for analysis were silicon treated. Analyses of these samples showed that the fiber lengths of SG-130 and RG-144 have similar mean and median values (Table II) with SG-130 (0.89 and 0.63) being slightly shorted than RG-144 (1.10 and 0.70). This can also be seen in comparison of their aspect ratios (Table V). Sample RG-244 SN has mean and median fiber lengths and aspect ratios which are longer than either those of SG-130 or RG-144, which can be seen in Tables II and V. It should be noted that the mode of RG-144 and RG-224 SN are the same (0.5 - 1.0) but the mode of SG-130 is lower than either of the other two original samples (0.0 - 0.5). The mode of each of the three original samples are lower than either their means or medians which indicates that all of these distributions are positively skewed. That is the largest component of each sample is smaller than either their mean or midpoint.

Another sample of RG-244 (RG-244 NS) was submitted, this one was not silicon treated. This sample was ultrasonicated and analyzed. The original sample of silicon treated RG-244 (RG-244 SS) was also ultrasonicated and reanalyzed. Table II and Table V show that these two samples were shorter than the original preparation of this sample. All three analyses of RG-244 are positively skewed, most of the fibers in the samples RG-244 SS and RG-244 NS were between aspect ratios 12 - 24 while sample RG-244 SN had its largest portion fall between 24 - 48.

In comparing the three different milled varieties of Chrysotile, sample RG-244 NS was used in comparison with SG-130 and RG-144 because it possessed closest the average values from all three RG-244 samples. Though shorter than the original values obtained for RG-244, sample RG-244 NS is still much longer than either SG-130 or RG-144 (See Tables II and V).

In comparing the finer portion of the Short Fiber Ore, the sample which was not ultrasonicated yielded closest to a standard distribution though it was also positively skewed with its largest component being in the range of 1.0 - 1.5 microns in length and 48 - 96 in aspect ratio. The ultrasonicated sample is shorter and is also positively skewed with its largest component falling in the range of 0.5 - 1.0 microns in length but with an aspect ratio of 48 - 96 as in the non-ultrasonicated sample (Tables II and V).

All samples had approximately the same results for fiber width distributions. The means ranged from 0.042μm to 0.055μm, the medians ranged from 0.040μm to 0.054μm and all samples had their largest component in the range from 0.03 - 0.05 microns (Table IV).

All sample were comprised of at least 75% fibers ≤ 5μm with SG-130 and RG-144 having the largest components at 100% and 99% respectively. The three RG-244 samples ranged from 75 to 89 percent with sample RG-244 SS having the greatest portions under 5μm followed sample RG-244 NS and RG-244 SN respectively (See Table III).

Discussion (continued):

The coarse portion of the short fiber ore is difficult to catagorize, the sample seems to be bi-modal with the primary mode of the length at 3.2 - 6.4 cm and the width at 0.0 - 0.1 cm. The secondary mode of the length distribution is at 0.0 - 0.1 cm and of the width at 0.8 - 1.6. The aspect ratio has one primary mode at 1.5 - 3. It should be noted that the course portion of the ore in its natural state is approximatly 64% non-fibrous. Only 36% of the course particulate of this sample has an aspect ratio of 3:1 or larger.

TABLE I

FIBER CLASSIFICATION CATEGORIES

CLASSIFICATION OF FIBERS WITH TUBULAR MORPHOLOGY

TM	-	Tubular Morphology, not sufficiently characteristic for classification as Chrysotile
CM	-	Characteristic Chrysotile Morphology
CD	-	Chrysotile SAED pattern
CQ	-	Chrysotile composition by Quantitative EDXA
CMQ	-	Chrysotile Morphology and composition by Quantitative EDXA
CDQ	-	Chrysotile SAED pattern and composition by Quantitative EDXA
NAM	-	Non-Asbestos Mineral

CLASSIFICATION OF FIBERS WITHOUT TUBULAR MORPHOLOGY

UF	-	Unidentified Fiber
AD	-	Amphibole by random orientation of SAED (shows layered pattern of 0.53 nm spacing)
AX	-	Amphibole by qualitative EDXA. The spectrum has elemental components that are consistent of an Amphibole.
ADX	-	Amphibole by random orientation SAED and qualitative EDXA
AQ	-	Amphibole by quantitative EDXA
AZ	-	Amphibole by one Zone Axis SAED pattern
ADQ	-	Amphibole by random orientation SAED and Quantitative EDXA
AZQ	-	Amphibole by one Zone Axis SAED pattern and Quantitative EDXA
AZZ	-	Amphibole by two Zone Axis SAED patterns with consistent inter-axial angle
AZZQ	-	Amphibole by two Zone Axis SAED patterns, with consistent inter-axial angle and Quantitative EDXA
NAM	-	Non-Asbestos Mineral

TABLE II

Central Tendencies - Length
(All Values In Microns)

Sample #	Mean	Median	Mode
SG-130	0.89	0.63	0.0 - 0.5
RG-144	1.10	0.70	0.5 - 1.0
RG-244 SS	2.45	1.51	0.5 - 1.0
RG-244 NS	2.86	1.25	0.5 - 1.0
RG-244 SN	3.22	1.90	0.5 - 1.0
Short Fiber Ore Not Treated	3.67	3.00	1.0 - 1.5
Short Fiber Ore Mortared and Ultrasonicated	2.89	1.75	0.5 - 1.0

RG-244 SS - This sample was silicon treated and ultrasonicated.
RG-244 NS - This sample was not silicon treated but was ultrasonicated.
RG-244 SN - This sample was silicon treated but not ultrasonicated.

TABLE III

Fiber Length Breakdown
(All Values Are Percent Of Total Fibers)

Sample #	Fibers ≤ 5μm	Fibers > 5μm and < 10μm	Fiber ≥ 10μm
SG-130	100	0	0
RG-144	99	1	0
RG-244 SS	89	9	2
RG-244 NS	84	11	5
RG-244 SN	75	18	7
Short Fiber Ore Not Treated	76	20	4
Short Fiber Ore Mortared and Ultrasonicated	82	15	3

RG-244 SS - This sample was silicon treated and ultrasonicated.
RG-244 NS - This sample was not silicon treated but was ultrasonicated.
RG-244 SN - This sample was silicon treated but not ultrasonicated.

TABLE IV

Central Tendencies - Width
(All Values In Microns)

Sample #	Mean	Median	Mode
SG-130	0.052	0.043	0.03 - 0.05
RG-144	0.054	0.043	0.03 - 0.05
RG-244 SS	0.055	0.043	0.03 - 0.05
RG-244 NS	0.054	0.054	0.03 - 0.05
RG-244 SN	0.043	0.040	0.03 - 0.05
Short Fiber Ore Not Treated	0.042	0.040	0.03 - 0.05
Short Fiber Ore Mortared and Ultrasonicated	0.046	0.040	0.03 - 0.05

RG-244 SS - This sample was silicon treated and ultrasonicated.
RG-244 NS - This sample was not silicon treated but was ultrasonicated.
RG-244 SN - This sample was silicon treated but not ultrasonicated.

TABLE V

Central Tendencies - Aspect Ratio
(All Values As Length:Width Ratio)

Sample #	Mean	Median	Mode
SG-130	17.3	12.6	12 - 24
RG-144	23.4	15.0	12 - 24
RG-244 SS	49.9	27.9	12 - 24
RG-244 NS	52.6	26.2	12 - 24
RG-244 SN	78.0	47.5	24 - 48
Short Fiber Ore Not Treated	91.8	75	48 - 96
Short Fiber Ore Mortared and Ultrasonicated	65.7	37.2	48 - 96

RG-244 SS - This sample was silicon treated and ultrasonicated.
RG-244 NS - This sample was not silicon treated but was ultrasonicated.
RG-244 SN - This sample was silicon treated but not ultrasonicated.

Transmission Electron Microscopy Analytical Report

Asteco Job Number: 8032
Report Date: November 30, 1991

Electron Optics Analysts:

Daniel R. Miller

Steig P. Brelloff

Barbara E. Lisk

G. David Browning

Laboratory Director:

Thomas J. Spangler

[redacted]

FIBER SIZE DETERMINATION

AND DISTRIBUTION OF

RG-244 CALIDRIA CHRYSOTILE ASBESTOS

BY

TRANSMISSION ELECTRON MICROSCOPY

for:

Client: KCAC, Inc.
P.O. Box K
King City, California 93930

Re: RG-244 Fiber Size Determination and Distribution

Job Number: 12515

Date: February 3, 1993

Analysts: N. J. Wiedemer
D. R. Miller

EXHIBIT
PIFX 19
11-18-94

Supervisor: T. J. Spengler

ASTECO, INC.
PO BOX 179
MIDDLEPORT, NEW YORK 14105
(716) 735-3894

[redacted]

OBJECTIVE:

To determine the fiber length and distribution of Chrysotile fibers in two unique samples of milled RG-244 Calidria Chrysotile asbestos, one treated with silicon, one untreated, by Transmission Electron Microscopy.

PROCEDURE:

The samples were prepared for analysis by water filtration and ultrasonic agitation in order to disperse the sample as completely as possible. A known amount of each sample was added to 500 ml of water and submersed in an ultrasonic bath for 30 minutes ~~minutes~~ to disperse the sample into suspension. A series of dilutions of each suspension was then filtered through 0.1μm pore size mixed cellulose esther filters and prepared for TEM analysis using a modified Jaffe wash and acetone condensation wash. The different dilutions of each sample suspension were then evaluated for fiber loading and preparation acceptability. Then one dilution of the untreated sample was chosen for analysis while all dilutions of the silicon treated samples were deemed unsuitable. The main problem evident in the treated sample was that the fibers clumped together and did not disperse sufficiently to allow measurement of individual fibrils.

The silicon treated sample was then resuspended in fresh water and ultrasonic agitation in excess of the original suspension was applied. The results of the second suspension of this sample did not improve significantly from the first. The process was repeated with extended ultrasonication only to produce similar results. The silicon treated sample was then resuspended a fourth time in a fresh 500 ml of water and placed in a mechanical shaker for slow agitation for 3 hours. The suspension was then immersed in an ultrasonicated bath for 30 minutes, after which dilutions were made and filtered. The preparations were evaluated and deemed to be slightly better than the previous three attempts and were used for the analysis of the silicon treated sample.

Analysis begun on the selected preparation and proceeded until 420 fibrils were counted. Asbestos fibers which normally would be counted as complex structures (bundles, clusters and matrices), were examined closely and the individual fibrils which composed the structure were counted and measured individually as accurately as possible. Complex structures which could not be accurately counted and measured as individual fibrils were discarded and not included in the fiber analysis (there were a great deal of complex structures discarded in the silicon treated sample). The lengths of the fibrils counted were measured at 20,000 magnification.

CALCULATIONS AND DEFINITIONS:

Fibers were distributed into different categories according to their lengths, for the purposes of this report a fiber is defined as a particle possessing parallel sides and an aspect ratio of 3:1 or greater. Aspect ratio is defined as the numerical proportion of length to width with the width being defined as 1. Although fiber widths were not measured, fibers were observed on screen and particles which appeared to be close to the 3:1 fiber limit were measured in both length and width dimensions to verify the classification of the particle.

CALCULATIONS AND DEFINITIONS (continued):

The mean, median and mode were also calculated for each sample. The mean used in this report is the arithmetic mean and is defined as :

$$\mu = \frac{\sum_{i=1}^{n} X_i}{n}$$

where μ = the arithmetic mean (length, width or aspect ratio)

n = the number of fibers counted

Xi= the quantity of the i^{th} fiber (length, width or aspect ratio)

i = the i^{th} fiber.

The median is defined as the middle variate for a population of variates arranged in either increasing or decreasing order for a population with an odd number of variates and the arithmetic mean of the two middle variates if the population has an even number of variates. The mode is the most frequently appearing variate in a population. In the report the mode is reported as an interval category of fiber lengths.

Positive Skewness is defined as a distribution in which the majority of the population is smaller than the mean, and the largest component is below the midpoint.

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All aspect ratios are referred to by the length or x component, thus a graph category of 3 - 6 would correspond to a range of aspect ratios from 3:1 to 6:1.

All length bar graph x values correspond to the midpoint of a length range. An x x value of 1.75 would refer to a length range of 1.5 μm to 2.0 μm.

An assortment of graphs are included to help illustrate sample distributions and various samples comparisons.

DISCUSSION:

The two sample, RG-244 silicon treated and Non silicon treated, show few major differences, this can be clearly seen in the Semi-Log Comparison of the two samples. The only difference is in percentage of total fibers made up by the smallest subdivision (0.0μm - 0.5μm), where there is clearly a greater percentage of the smallest fibers present in the untreated sample. The percentage of fibers ≤5μm are comparable (89% in the silica treated sample and 92% in the untreated sample) and the largest division (≥10μm) is identical in both samples.

The mean, and median fiber length of the untreated sample (1.83μm, 1.00μm) is noticeably lower than that of the silicon treated sample (2.22μm, 1.25μm) and while the mode for each sample was different, the untreated sample possessed almost equal separation between the length categories 0.0μm - 0.5μm and 0.5μm - 1.0μm which could possibly be considered to be one larger mode (0.0μm - 1.0μm) as a combination of the two smaller categories.

All samples were positivly skewed.

QUALITY ASSURANCE:

ASTECO is accredited by the American Industrial Hygiene Association for the analysis of asbestos. Our accreditation number is 356. This accreditation may be verified by contacting the American Industrial Hygiene Association at (216) 762-7294. The laboratory is also accredited by the New York State Environmental Laboratory Approval Program (ELAP) for the analysis of fibers and asbestos. Our accreditation number is 10834. This accreditation may be verified by contacting ELAP at 518 474-4471. The laboratory has accreditation by NIST (National Institute of Standards and Testing) for the NVLAP program (National Voluntary Laboratory Accreditation Program). This accreditation may be verified by contacting NIST at (301) 975-4016.

LIMITS AND CONDITIONS

1. In compiling this report ASTECO INC. (hereinafter We/Our) has followed procedures which we believe to be accurate and reliable but there may be other methods for evaluating asbestiform minerals which may produce different qualitative results.

2. The analytical results reported herein are not to be viewed as exactly equal to the results which may be found at different sampling locations due to the nonuniformity in concentration of these types of substances.

3. Any use of, or reliance upon, the data, or other information, contained herein by yourself, or any other party, shall be without any recourse to, or liability on Our part.

4. The breathing of asbestos dust may cause serious bodily harm and We can make no warranty or guarantee that the results contained herein indicate that safe levels of exposure exist. It is the responsibility of any user of asbestos or an asbestos-containing product to ensure that safe handling procedures are employed and that applicable government regulations are complied with.

TABLE 1

FIBER CLASSIFICATION CATEGORIES

CLASSIFICATION OF FIBERS WITH TUBULAR MORPHOLOGY

TM - Tubular Morphology, not sufficiently characteristic for classification as Chrysotile

CM - Characteristic Chrysotile Morphology

CD - Chrysotile SAED pattern

CQ - Chrysotile composition by Quantitative EDXA

CMQ - Chrysotile Morphology and composition by Quantitative EDXA

CDQ - Chrysotile SAED pattern and composition by Quantitative EDXA

NAM - Non-Asbestos Mineral

CLASSIFICATION OF FIBERS WITHOUT TUBULAR MORPHOLOGY

UF - Unidentified Fiber

AD - Amphibole by random orientation of SAED (shows layered pattern of 0.53 nm spacing)

AX - Amphibole by qualitative EDXA. The spectrum has elemental components that are consistent of an Amphibole.

ADX - Amphibole by random orientation SAED and qualitative EDXA

AQ - Amphibole by quantitative EDXA

AZ - Amphibole by one Zone Axis SAED pattern

ADQ - Amphibole by random orientation SAED and Quantitative EDXA

AZQ - Amphibole by one Zone Axis SAED pattern and Quantitative EDXA

AZZ - Amphibole by two Zone Axis SAED patterns with consistent inter-axial angle

AZZQ - Amphibole by two Zone Axis SAED patterns, with consistent inter-axial angle and Quantitative EDXA

NAM - Non-Asbestos Mineral

TABLE II

Central Tendencies - Length
(All Values In Microns)

Sample #	Mean	Median	Mode
RG-244 S	2.22	1.25	0.5 - 1.0
RG-244 N	1.83	1.00	0.0 - 0.5

TABLE III

Fiber Length Breakdown
(All Values Are Percent Of Total Fibers)

Sample #	Fibers ≤ 5μm	Fibers > 5μm and < 10μm	Fiber ≥ 10μm
RG-244 S	89	9	2
RG-244 N	92	6	2

RG-244 S - Sample was treated with silicon.
RG-244 N - Sample was not treated with silicon.

Transmission Electron Microscopy Analytical Report

Asteco Job Number: 12515
Report Date: February 3, 1993

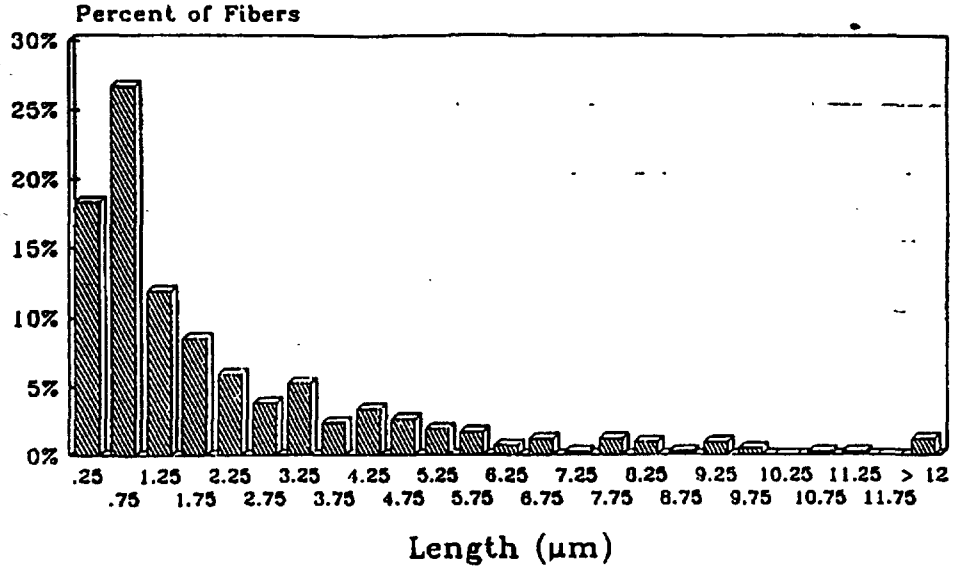
Electron Optics Analysts: Noelle J. Wiedemer

Daniel R. Miller

Laboratory Director: Thomas J. Spengler

GRAPHS
Fiber Size Distributions

Fiber Length Distribution
RG-244



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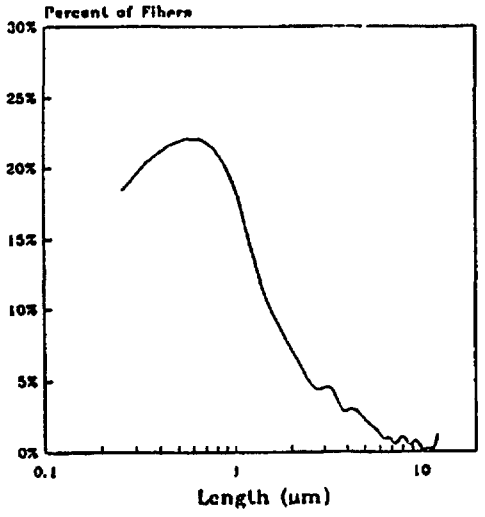

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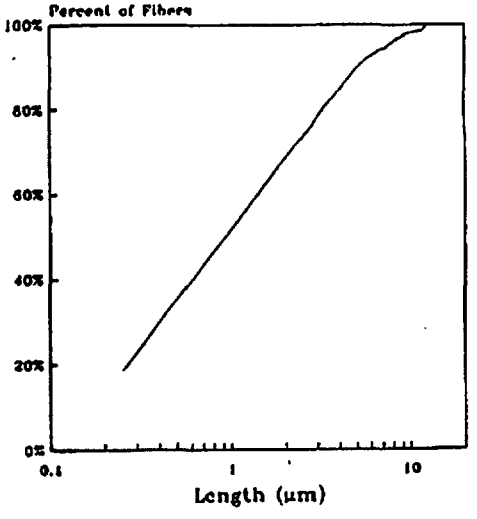

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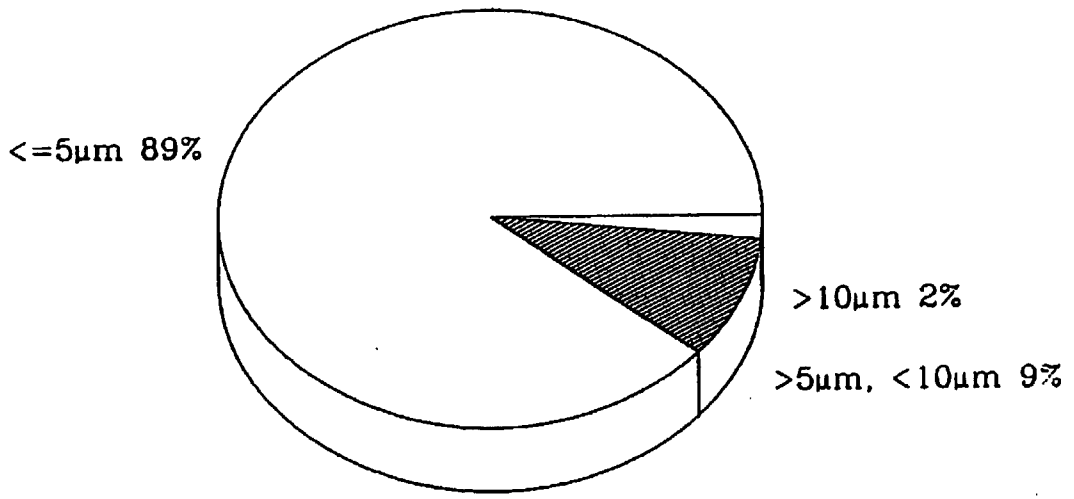

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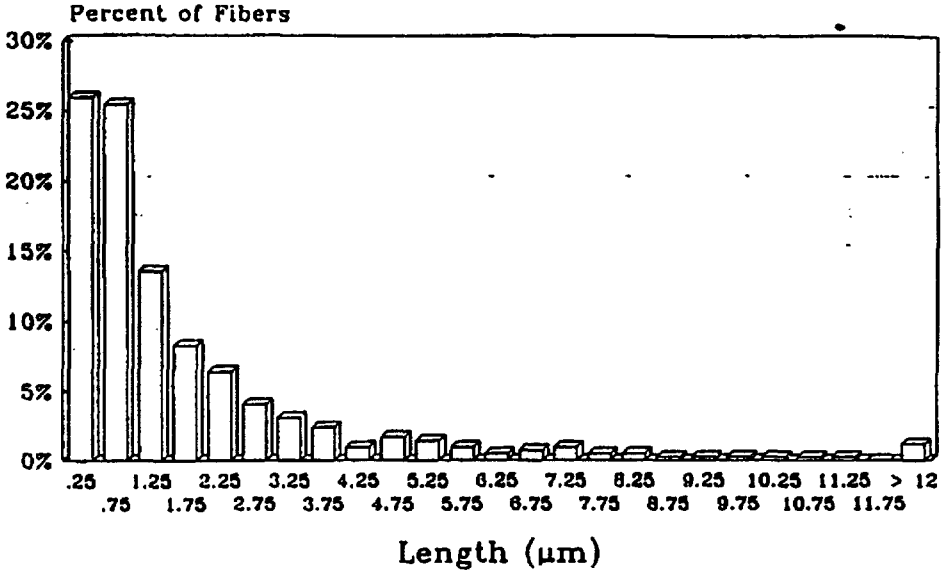

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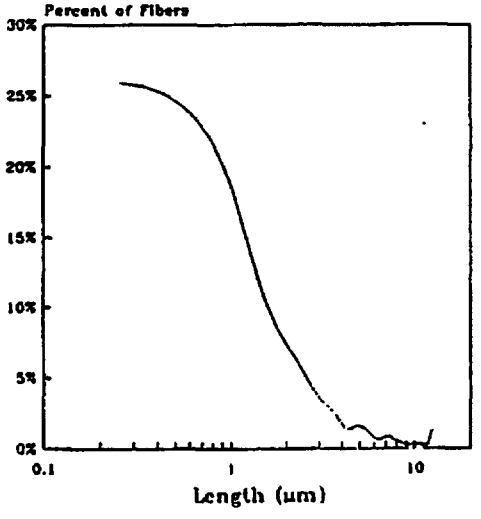

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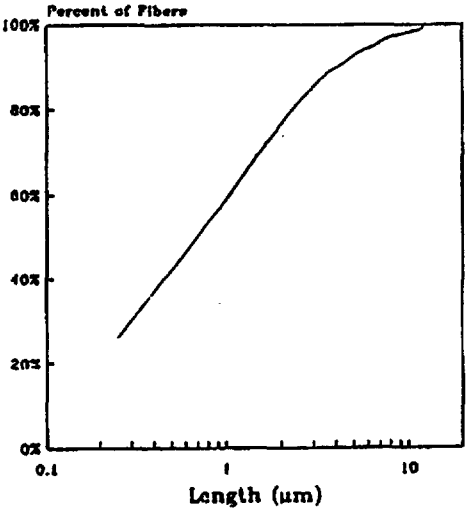

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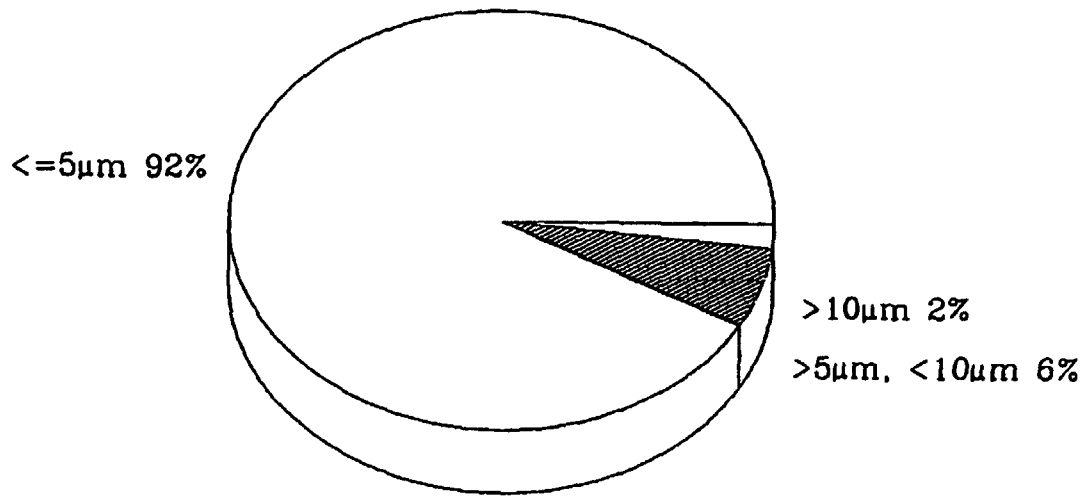

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SIZE DISTRIBUTIONS OF OCCUPATIONAL AIRBORNE ASBESTOS TEXTILE FIBRES AS DETERMINED BY TRANSMISSION ELECTRON MICROSCOPY

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Abstract—The size distributions of airborne chrysotile fibres, associated with the processes of carding, spinning and weaving in an asbestos textile factory, have been evaluated by transmission electron microscopy. The distributions were approximately log-normal, with little change between processes. Median lengths and diameters were 1.6 and 0.08 μm, respectively. The results indicate that about 60% of fibres would not be seen by scanning electron microscopy, under the usual conditions, and an even larger proportion would be missed by optical microscopy. For fibres longer than 5 μm, then, about 70% of fibres would be seen by the scanning electron microscope. An optical microscope with a resolution of 0.3 μm would see approximately one quarter of all fibres greater than this length.

INTRODUCTION

TRANSMISSION electron microscopy (TEM) allows the observation of even the smallest asbestos fibre (the chrysotile fibril), which can have a diameter as low as 0.01 μm (GIBBS and HWANG, 1975). The complete fibre size distribution of a sample of asbestos may therefore be evaluated by TEM, in contrast to scanning electron microscopy (SEM) and optical microscopy, which in practice can only detect diameters much larger than the smallest asbestos fibres. TEM-derived size distribution data for asbestos manufacturing industries are rather scarce in the literature, probably because the analyses are expensive compared with those by the other techniques. This paper reports TEM measurements of airborne chrysotile fibres in an asbestos textile factory.

Current monitoring of airborne asbestos dust in factories (HEALTH AND SAFETY EXECUTIVE, 1983) involve collecting the dust on membrane filters and counting fibres longer than 5 μm, according to certain rules, under a phase-contrast optical microscope (PCOM). The detection limit of a good, correctly adjusted PCOM may be 0.15 μm (ROOKER *et al.*, 1982); however, under normal operating conditions, the detection limit is not so low (HWANG and WANG, 1983; ASHCROFT and HEPPLESTON, 1973; HWANG and GIBBS, 1981) and we shall assume a limit of 0.3 μm.

Earlier work by GIBBS and HWANG (1975), using SEM, found that chrysotile fibres tended to be smaller in both length and diameter at later stages of processing. For carding in a textile plant, the median length and diameter were 1.0 and 0.15 μm, respectively; 2.3% of fibres were longer than 5 μm. However, there is a possibility that some fine fibres were lost in sample preparation (WALTON, 1982). Moreover, because of

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the SEM's detection limit, fibres thinner than 0.1 μm were unlikely to be seen (Middleton, 1982).

Dement and Harris (1979) report several TEM-derived fibre size distributions for asbestos. We calculated the following from their data: in the chrysotile-using textile plant, for the processes of fibre preparation, twisting and weaving, the geometric mean lengths were 2.2, 1.8 and 2.0 μm, respectively, and the geometric mean diameters were 0.17, 0.14 and 0.14 μm, with 41%, 57% and 58% thinner than 0.1 μm; 85%, 84% and 78% thinner than 0.3 μm; and 11%, 10% and 11% longer than 5 μm. Hwang (1983) has reported TEM distributions for chrysotile mining and milling; his diameters and lengths were considerably smaller.

METHOD

Static samples, collected on either Gelman DM Metricel DM450 or DM800 filters (25 mm diameter), at flow rates of 0.4 and 1.5 l. min^{-1}, respectively, were taken within about 2 m of machines and the extraction systems associated with the processes of carding, spinning and weaving. Sampling times of 60 and 120 min were used.

The filters were mounted onto nickel, 200-mesh high-transmission TEM grids, using the direct transfer technique (Burdett, 1983). Analysis was carried out on a Philips EM400T analytical transmission electron microscope, equipped for energy-dispersive X-ray analysis (EDXA) and for scanning (transmission) electron microscopy (STEM).

The grid was first inspected at low magnification for an even distribution of deposit: if it appeared uneven, the sample was rejected.

Only chrysotile was to be included in the study; chrysotile fibres were identified by their characteristic EDXA spectrum and morphology (Carton and Kaufer, 1980). A note of any other fibres found was made so that they would not be included at a later stage.

A fibre has in the past been defined, by the Health and Safety Executive, as a particle having an aspect ratio greater than three and having parallel sides for most of its length.

Crossed fibres, fibre bundles, and piles of fibres were selected for measurement using the rules adopted in France (Carton and Kaufer, 1980); these rules are similar to those in the European Directive method (Health and Safety Executive, 1983), except for the requirement concerning parallel sides, and that fibres are not rejected on the grounds of attachment to non-fibrous particles. Only fibres with diameters less than 3 μm, that is, respirable fibres, were included.

The unit counting area was one grid square of 55 μm side length. Between two and ten randomly selected squares were analysed, such that, except for samples of very low fibre density, a minimum of 100 fibres per grid were counted. Only those fibres wholly in the counting area and those crossing either the upper side or the left side and ending in the field were included (Asbestosis Research Council, 1971).

A micrograph of each complete counting area was taken for reference. The fibres in each area were then photographed at the higher magnification of 3600. Since the boundaries of the counting area were the electron-opaque grid bars, any fibres that crossed one of the counting sides were photographed on the STEM facility in the secondary electron imaging mode (various magnifications used).

Fibre measurements were made using a Reichert-Jung 'Videoplan' semi-automatic

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nage analysis system, calibrated using a micrograph of a standard cross grating (2160 lines mm^{-1}), also taken at 3600 magnification: the micrograph negatives were enlarged by a factor of 9.5 by projection onto a measuring tablet, and the true fibre length (the length of the fibre after straightening) and the true fibre diameter (the width of a central portion of the fibre) (GIBBS and HWANG, 1975) were measured using a cursor, anti-parallax cross-hairs, and a ×7 magnifying eyepiece, giving edge measurements to an accuracy of 3 nm.

The aspect ratio (true length/true diameter) was calculated for each fibre.

RESULTS AND DISCUSSION

Fourteen samples were analysed: four from the spinning process, from which 424 respirable fibres were measured, and five samples from each of carding and weaving, from which 519 and 495 fibres, respectively, were measured. Some non-chrysotile fibres were observed but not included in the study.

Means and standard deviations, both arithmetic and geometric, and the median values for each of the parameters, true fibre length, true diameter and aspect ratio, for the three processes, carding, spinning and weaving, are summarized in Table 1. Figures 1 to 3 show the cumulative frequency distributions for each process.

Figure 4 summarizes the individual sample details: these graphs show that there was little variation between samples.

The frequency distributions were approximately log-normal. Comparison of the distributions shows that there was little variation between processes. The group means,

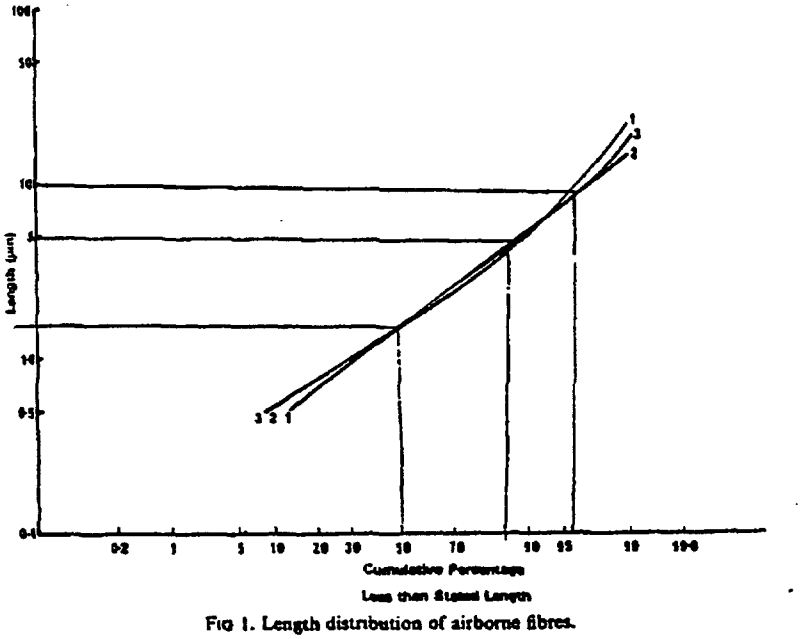

FIG 1. Length distribution of airborne fibres.

TABLE I. SUMMARY TABLE OF AIRBORNE FIBRE PARAMETERS

Process	Length (μm)					Diameter (μm)					Aspect ratio				
	Arit. Mean	Arit. S.D.	Median ⊥	Geom. Mean	Geom. S.D.	Arit. Mean	Arit. S.D.	Median ⊥	Geom. Mean	Geom. S.D.	Arit. Mean	Arit. S.D.	Median	Geom. Mean	Geom. S.D.
Carding	2.8	3.2	1.6	1.5	2.6	0.14	0.18	0.08	0.09	2.4	26	33	17	18	2.2
Spinning	2.5	2.7	1.6	1.7	2.3	0.11	0.15	0.07	0.08	2.3	30	26	22	22	2.2
Weaving	2.7	3.0	1.6	1.7	2.4	0.13	0.20	0.08	0.08	2.3	28	31	19	20	2.2

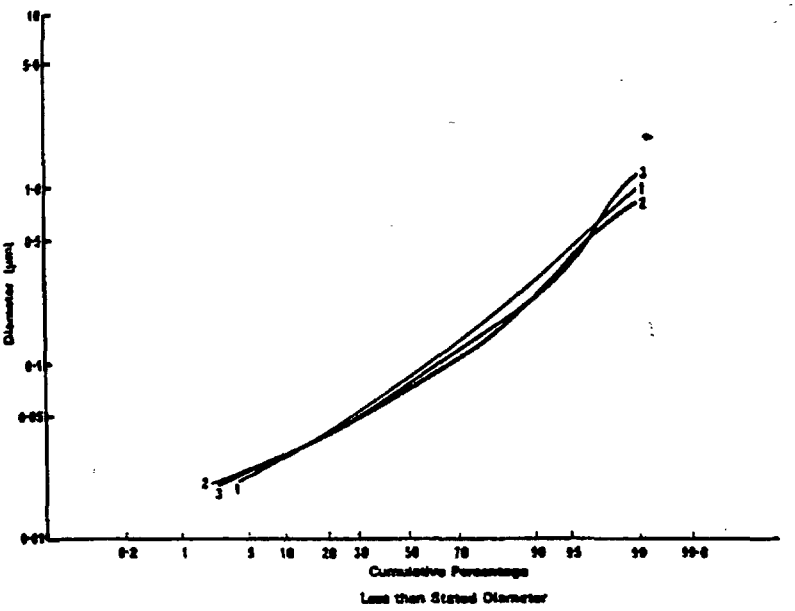

FIG. 2. Diameter distribution of airborne fibres.

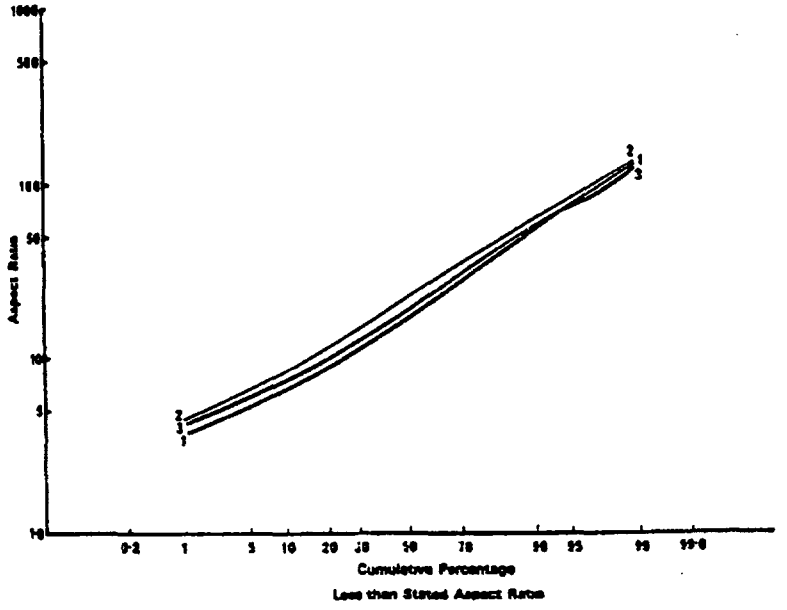

FIG. 3. Aspect ratio distribution for airborne fibres.

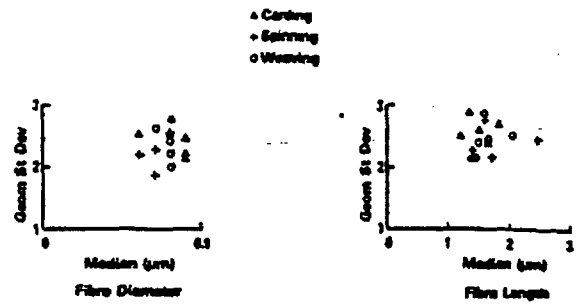

FIG. 4. Individual sample results.

TABLE 2. PROPORTION OF RESPIRABLE FIBRES OF (a) ALL LENGTHS AND (b) LENGTHS > μm, AS A FUNCTION OF DIAMETER

(a) Process	Percentage of fibres (all lengths) with diameter greater than (μm)			
	0.1	0.2	0.3	0.4
Carding	41	17	8.3	5.4
Spinning	35	11	7.8	4.9
Weaving	37	13	6.9	4.7

(b) Process	Percentage of fibres (lengths > 5 μm) with diameter greater than (μm)			
	0.1	0.2	0.3	0.4
Carding	77	49	26	20
Spinning	69	31	19	14
Weaving	78	38	26	24

standard deviations and medians for each of the three parameters also show close agreement.

For all respirable fibres, the geometric mean diameter for carding was 0.09 μm and for spinning and weaving was 0.08 μm. About 60% of all fibres had diameters under 0.1 μm (the SEM detection limit) and 93% were under 0.3 μm (the optical limit) (Table 2a).

The geometric mean length for carding was 1.5 μm and for spinning and weaving was 1.7 μm. About 11% were longer than 5 μm; of these, about 74% had diameters exceeding 0.1 μm and 25% exceeding 0.3 μm (Table 2b).

The fibres found in the factory visited for our study tended to be thicker and slightly longer than those from the factory of DEMENT and HARRIS (1979). In neither study were there any substantial differences in size distributions of the various processes in the same factory.

Airborne size distributions may be a function of the fibre characteristics, such as grade and origin, rather than the processing equipment.

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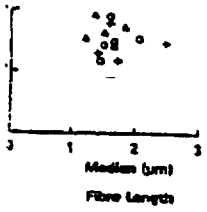

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CONCLUSIONS

(a) There was not much difference in the size distribution of respirable fibres between individual samples and processes.

(b) The distributions were approximately log-normal, with a median length of 1.6 μm (geometric standard deviation 2.4) and a median diameter of 0.08 μm (2.3).

(c) About 60% of all fibres had sizes below the detection limit of the usual SEM method and about 93% had sizes below the detection limit of the PCOM.

(d) For fibres longer than 5 μm, then, about 26% of fibres were of sizes below the detection limit of the usual SEM technique and 75% had sizes below the detection limit of the PCOM.

NB

Acknowledgement—We would like to thank Dr S. J. Rooker for the samples used in this work.

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Characterization of Three Types of Chrysotile Asbestos after Aerosolization

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Jeffrey Mine and Coalinga Mine chrysotile, two asbestos samples prepared for experimental research by the National Institute of Environmental Health Sciences, and the UICC B chrysotile reference sample have been characterized in the aerosolized state using gravimetric measurements, light microscopy, scanning electron microscopy, and x-ray energy spectrometry. These methods revealed (1) a greater "respirable" mass fraction in the Jeffrey and UICC B preparations compared to the Coalinga sample, (2) for fibers greater than 5 μm in length and less than 3 μm in diameter, Jeffrey Mine chrysotile contained a significantly greater fraction of fibers longer than 40 μm in length compared to the UICC B or Coalinga Mine chrysotiles, and (3) Jeffrey and UICC B chrysotile contained no fibers or fiber clusters which exceeded 2 μm in diameter while Coalinga chrysotile contained numerous fibers and fiber clusters which were greater than 2 μm in diameter. The characterization of these chrysotile preparations in the aerosolized state, in particular the Coalinga Mine chrysotile, demonstrated different fiber length and fiber width distributions when compared with previous characterizations of samples that had been dispersed in a liquid medium by ultrasonification. These observations emphasize the importance of determining the size distribution of fibers in the aerosolized state for inhalation studies and the size distribution of fibers in a liquid suspension for oral ingestion, instillation, or injection studies. Because of differences in length–width distributions, each of the studied chrysotile preparations would be expected to have different patterns of deposition in the alveolar regions of the lung after an inhalation exposure.

INTRODUCTION

There is increasing evidence that asbestos-induced pulmonary fibrosis and neoplasia are due to the physical and chemical characteristics of the inhaled fibers (Wagner, 1965; Seaton, 1975; Stanton and Layard, 1978; Pott, 1978). This makes it important to use well-characterized fiber preparations in experimental research to more clearly identify those factors leading to lung injury. The purpose of this paper is to describe two new samples of chrysotile which have been prepared for experimental research by the National Institute of Environmental Health Sciences (NIEHS). A third chrysotile, UICC B, has also been characterized in this paper.

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Chrysotile represents over 90% of the world's asbestos production and is ubiquitous in our environment. The properties of fire retardance, chemical inertness, tensile strength, and flexibility make chrysotile important in numerous applications such as insulation, ceiling tiles, brake linings, and cement products. These utilizations of asbestos enhance the frequency of inhalation of chrysotile by people not directly involved with the mining or processing of this material. Chrysotile is a member of the serpentine family of minerals, whereas all other asbestos minerals belong to the amphibole family (Sinclair, 1959). Mechanical agitation of chrysotile can lead to disruption of the fiber at weak points along the fiber. This disruption can cause the fiber to "open up" into its fibrillar subunits, creating new fibers of smaller diameter and/or shorter length, fibers with splayed ends, fibers with an uneven diameter along the length, or combinations of the above (Assuncao and Corn, 1975). These features make chrysotile more complex to analyze in terms of fiber size and number than the amphibole types of asbestos in which fibers are basically straight and uniform in diameter.

The characterization of a chrysotile preparation in terms of particle and fiber size distribution is influenced by a number of factors. These include (1) the state in which the chrysotile is found, i.e., in bulk, in suspension, or in an aerosol, (2) the method of sample collection, i.e., on a slide or filter, (3) the manner in which the collected sample is prepared for examination, i.e., by ultrasonification, transfer or direct preparation of the filter or slide, (4) the instrument used to measure the particles and fibers in the sample, i.e., the optical microscope, transmission electron microscope, or scanning electron microscope, and (5) the criteria used for defining fibers, i.e., a minimum fiber length, a 3 to 1 aspect ratio, or characteristics of fiber morphology.

Characterization of chrysotile in the aerosolized state has been done for the UICC A and B reference samples (Timbrell, 1970a; Beckett, 1973). Two new research samples of chrysotile have been prepared in bulk (approximately 1000 pounds of each) and are available for experimental research through the National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina. These new preparations (Jeffrey and Coalinga chrysotile) have been characterized for elemental composition, mineral composition, particle surface area and density, and thermal properties using optical emission spectrography, x-ray diffraction, thermogravimetry, pycnometry, and a number of petrographic microscopic techniques (Campbell *et al.*, 1980). Particle size analysis has been done with samples dispersed in a liquid medium in which measurements of fiber length and diameter were made (Wylie, 1979; Campbell *et al.*, 1980; Siegrist and Wylie, 1980). Particle size analysis of these preparations in the aerosolized state was not done. The purpose of this study is to characterize in the aerosolized state the Jeffrey and Coalinga chrysotile preparations along with the UICC B reference sample. To characterize each preparation, gravimetric dust measurements, optical microscopy, scanning electron microscopy (SEM), and x-ray energy spectrometry were used. Information obtained using these techniques has helped identify characteristics of these asbestos preparations which may influence their deposition pattern when inhaled as an aerosol and which could potentially contribute to their ability to cause pulmonary injury.

MATERIALS AND METHODS

Fiber Preparations

Chrysotile samples were obtained from the following locations: the Coalinga Mine in California (Union Carbide), the Jeffrey Mine in Quebec, Canada (Johns–Manville), and the Canadian reference sample prepared by the International Union Against Cancer (UICC B). A brief history for each preparation is given.

Coalinga Mine fiber. Identified as COF-25, this unique form of chrysotile is obtained from the New Idria serpentinite mass located in the Diablo range in California. The deposit is unusual in that the fibers are randomly oriented as a mat, rather than as parallel fibers running in veins, and the mining process is done with bulldozers. The deposit is almost pure chrysotile. The fibers are short in length and are not of spinning grade. Because of the absence of long fibers in this chrysotile, a great deal of interest has been generated in using this chrysotile as a "short-range fiber" preparation.

Preparation of this short fiber material for experimental research was done in the following manner. The ore from the mine was first screened to remove contaminating rocks. From this point the material was processed in water as a slurry of 1% solids and 99% water. The slurry was passed through a grinder and a magnetic separator three times to open the fiber bundles and to remove any iron-containing minerals. Between each grinding the slurry was passed through a particle size separator (hydroclone) under pressure. The aqueous slurry was rotated inside the hydroclone forming a vortex. Heavy particles escape from the hydroclone through a side port located near the bottom of the hydroclone. These coarse particles were reground and put through the hydroclone again. The light particles leave the hydrocione through a side port located near the top of the vortex. These particles were fed into a series of hydroclones in which the top exit ports (overflow port) are progressively smaller to allow for the collection of finer and finer chrysotile fibers. The final hydroclone port had an external diameter of 25 mm from which the chrysotile preparation was collected. (For industrial purposes the final hydroclone port is usually six inches (personal communication, Asbestos Group, Union Carbide, Niagara Falls, N.Y.).)

Langer *et al.* (1978) described in detail a chrysotile sample also obtained from the Coalinga Mine deposit, referred to as Calidria RG-144. The differences between COF-25 and RG-144 are a result of the differences in the processing of the raw material. COF-25 has a finer particle size than RG-144. This finer particle size is a result of differences in the pressure, vortex characteristics, and overflow port diameters used in the hydroclone. COF-25 is not derived from RG-144 by further grinding or pellet milling.

Jeffrey Mine fiber. Identified as Plastibest-20, this form of chrysotile is a grade 4 asbestos used by the plastics industry. The fibers run in parallel bundles, oriented crosswise in veins in serpentine rock and for this project were purified by roller milling and air separation using standard industrial techniques. The final fiber preparation by volume is greater than 96% chrysotile (Campbell *et al.*, 1980). Preparation of the material for experimental research was accomplished by passing the material through a hurricane pulverizer three times to open fiber bundles.

UICC B chrysotile reference sample. This preparation is a grade 4 chrysotile (Timbrell *et al.*, 1968) obtained from eight different Canadian chrysotile mines and mixed according to the proportional production of each mine during the year of 1950. The approval of this preparation was made in 1966 by the Union Internationale Contre Cancer (UICC) to standardize asbestos samples used in experimental research. The literature is replete with information regarding the physical and chemical makeup of this chrysotile preparation (Timbrell *et al.*, 1968; Timbrell, 1970a, b; Rendall, 1970, 1980; Morgan and Cralley, 1973; Beckett, 1973). We have reexamined UICC B chrysotile in the aerosolized state with a twofold purpose: (1) to compare our results for the UICC B fiber size distribution with those found in prior studies in the literature, and (2) to correlate the size distribution of the Coalinga and Jeffrey Mine fibers to the UICC chrysotile fiber preparations available for experimental inhalation research. No manipulation of these chrysotile preparations was done prior to aerosolizing the fibers.

Fiber Aerosolization

A modified Timbrell generator (Timbrell, 1968) was used to create a dust cloud within a 5-m^3-stainless steel-exposure chamber. Each asbestos preparation was hand compressed with a plunger in a 2.8 × 9.0-cm cylinder to form a plug. This was mechanically advanced into the pathway of a blade rotating 1500 rpm to create an aerosol of fibers within the dispersing bowl of the generator. A copper tube connecting the dispersing bowl to the exposure chamber facilitated the passage of aerosolized fibers into the exposure chamber. The concentration of the asbestos dust cloud was regulated by adjustment of the airflow through the exposure chamber. This resulted in the flow of air being maintained between 200 to 400 liters per minute.

Total Dust Mass Concentration

Once the exposure chamber had stabilized (normally after 1 hr of dust generation), a gravimetric measurement of the dust concentration within the exposure chamber was made. This was accomplished by sampling 100 liters of chamber air drawn through a 0.8-μm Nucleopore filter housed within a Gelman filter holder. The sample time was 10 min at a flowrate of 10.0 liters per minute.

Respirable Mass Concentration

The respirable mass concentration in the exposure chamber for each chrysotile preparation was measured using two different instruments: a Casella sampler and a Cascade impactor. The term "respirable mass concentration" is arbitrary in that it was decided by totally different parameters for each apparatus used. Using the Casella sampler, a gravimetric estimate of the respiratory mass concentration was made by collecting fibers and particles with an equivalent aerodynamic diameter of 7.1 μm or less on a glass fiber filter. Fibers and particles larger than this equivalent diameter were prevented from depositing on the filter by a multichannel horizontal elutriator. Each sample was collected over a 6-hr period at a flowrate of 2.5 liters per minute with the Casella sampler placed inside the exposure chamber.

Using the Cascade impactor, the respirable mass concentration was defined to

include filters on which the majority (>50%) of fibers collected were less than 10 μm in length. This was determined by examination of the filter by optical microscopy. This respirable mass was obtained by using precutter stages in the Cascade impactor to eliminate the longer and thicker fibers and by adjusting the flowrate to obtain a fiber size distribution (cut point) of 10 μm or less in length.

The volume of air sampled through the Cascade impactor was 200 liters. A total sample mass was also collected on the same day by sampling 0.2 m^3 of chamber air at a flowrate of 10.5 liters per minute for 19 min 3 sec using the same setup minus the Cascade impactor. All gravimetric measurements were expressed in mg/m^3. Gravimetric measurements of the respirable mass collected in the Cascade impactor were done for the Jeffrey and Coalinga Mine preparations only, because of the known similarity between Jeffrey and UICC B chrysotile.

Collection of Fiber Samples

The same apparatus used to collect samples for dust concentration measurements was also used to collect fiber samples. Samples to be analyzed by light microscopy were collected on Millipore-type AAWP membrane filters at a flowrate of 0.1 liters per minute for 3 to 5 min depending upon the chamber concentration of the chrysotile preparation. Before use, the filters were treated in a 0.1% solution of Hyamin 2389 and dried at room temperature overnight to prevent static charging of the filter. Samples analyzed by scanning electron microscopy were collected on a 0.2-μm Nucleopore filter for 2 sec and 5 sec at a flowrate of 10 liters per minute.

Preparation of Filters for Examination

Light microscopy. After sampling, the filter was placed on a 25 × 75-mm glass microscope slide, sample side down. The filter was cleared by holding the slide over an evaporating flask of boiling acetone. After clearing, a drop of glycerol-triacetate (Permount) was placed on the dissolved filter and a cover slip applied.

Electron microscopy. Filters were secured to the polished side of a Gough planchet with carbon paint and were gold coated. The thickness of the gold coat was 100 to 150 nm.

Fiber Characterization

Light microscopy. A Beckett G22 graticule was used. At 500×, the magnification used for sizing and counting, the graticule was a square, 100 μm on each side. The lattice on the graticule had a spacing of 5 μm in one direction and 3 μm in the perpendicular direction. The following rules were used for fiber characterization: All fibers counted had at least a 3 to 1 length-to-diameter ratio (aspect ratio). Only fibers 5 μm in length or longer were counted. A fiber bundle which met the 3 to 1 aspect ratio requirement was counted as a fiber. Bundles with a diameter greater than 3 μm were not counted. Only fibers whose midpoint was located within the graticule were counted. At least 200 fibers were counted from 20 to 100 random graticule fields for each filter sample. The characterization of each chrysotile preparation by light microscopy was based upon measurements from filters collected on a daily basis (5 days/week for 12 months).

Electron microscopy. The magnification used for particle sizing and counting

was 10,000. To facilitate measurement of long fibers and fiber width, magnifications ranging from 1200 to 18,000× were used. Counting and sizing of particles were done directly on the electron microscope screen. Only particles whose midpoint fell within the area of the viewing screen were analyzed. This procedure essentially reduced all particles to points, thus causing every particle to have an equal probability of being counted. To assure accuracy in measurement, a standard was made consisting of latex beads 1.099 μm in diameter on a 0.2-μm Nucleopore filter. The filter and beads were gold coated and used to verify the magnification prior to each counting session. The area of the filter analyzed consisted of bands randomly selected across portions of the filter. A field was randomly selected at 10,000 magnification and then moved to the right. Several bands were counted per filter and a range of 4 to 8 filters was analyzed for each chrysotile preparation.

The following three categories were used to characterize the material on the filters for each of the chrysotile preparations:

(1) Fiber: (a) At least a 3 to 1 aspect ratio was required. (b) Generally, a fiber was cylinder-shaped with a uniform diameter. However, a slight separation of the fibrils along any section of the fiber or at the ends was permissible. (c) The diameter of the fiber was measured at the widest portion of the fiber whether it occurred at the end or at some point along the fiber. (d) Smaller fibrils solidly attached to the major body of the fiber constituted a portion of the fiber. It was not counted as a separate fiber. The diameter of the fiber would be measured at this point if it constituted the widest portion of the fiber.

(2) Fiber cluster: (a) A fiber mass with at least a 3 to 1 aspect ratio was required. (b) It was composed of small fibrils usually oriented in the same direction, but partially separated. Separation of fibrils may occur at any point along the length of the bundle or cluster. (c) The diameter of the cluster was measured at its greatest combined width along the fiber mass. A fiber cluster usually has a more variable width than a "fiber."

(3) Nonfibrous particle: (a) Any particle with less than a 3 to 1 aspect ratio. (b) The longest dimension was measured as well as the greatest perpendicular measurement.

Fiber "flocs" or clumps were also occasionally present on the filter consisting of tangled fibers and fiber clusters. These were not analyzed because their complexity prevented accurate separation into individual fibers and fiber clusters.

Representative fibers, clusters, and nonfibrous particles were analyzed for elemental composition using x-ray energy spectrometry for each chrysotile preparation. Each particle was analyzed for the presence of an element by a scoring system ranging from 0 to 4+. At 10,000×, 50 nonfibrous particles whose midpoints fell within the viewing area of a randomly selected band across the filter were analyzed. In addition, the magnesium-to-silicon ratio was determined for all particles containing these two elements.

RESULTS

Dust Concentration Measurements

The total mass concentration and corresponding respirable mass concentration for each chrysotile preparation are given in Table 1 using both the Casella sampler

TABLE 1
GRAVIMETRIC MEASUREMENTS FOR EACH CHRYSOTILE PREPARATION IN THE EXPOSURE CHAMBER

Preparation	(A) Chamber dust mass concentration (mg/m^3)[a]	(B) Respirable concentration (mg/m^3)[a]	Ratio B/A
		Casella sampler	
Jeffrey	11.36 ± 2.18	9.90 ± 1.63	0.871
UICC B	10.99 ± 2.11	8.32 ± 1.75	0.757
Coalinga	7.76 ± 1.46	3.28 ± 0.83	0.423
		Cascade impactor	
Jeffrey	12.29 ± 3.33[b]	2.62 ± 0.81[b]	0.213
Coalinga	15.63 ± 2.41[c]	0.80[c]	0.051

[a] All data are means ± SD, $n = 240$ for each chrysotile preparation.
[b] For Jeffrey all data are means ± SD, $n = 12$.
[c] For Coalinga the chamber mass concentration is based on three samples and the respirable concentration is a single sample.

and the Cascade impactor. When comparing the ratio of the respirable mass concentration to the total dust concentration found in the exposure chamber, the Jeffrey Mine chrysotile and UICC B chrysotile have a relatively high percentage of respirable material based upon the Casella sampler measurements (87% for the Jeffrey chrysotile and 76% for the UICC B chrysotile). The ratio of the respirable mass concentration to the total mass concentration is significantly lower for Coalinga Mine chrysotile (42%). Using the Cascade impactor a substantially smaller fraction of the total dust concentration was found to be respirable. For the Jeffrey fiber 21% of the total dust concentration was respirable and 5% of the total dust concentration was respirable for the Coalinga fiber.

Fiber Characterization—Light Microscopy

Table 2 lists the percentage of fibers found for each given length interval by light microscopy for each of the three chrysotile preparations. Fibers greater than 100 μm in length and less than 3 μm in diameter were found in each preparation. The percentage of fibers present from 5 to 30 μm in length was similar for all three aerosolized preparations with greater than 70% of all counted fibers falling within this length interval. For the fiber-length intervals of 40–50, 50–100, and greater than 100 μm, the percentage of fibers contained within each of these length intervals was significantly greater ($P < 0.05$) for the Jeffrey Mine preparation than for either the UICC B preparation or the Coalinga Mine preparation.

Fiber Characterization—SEM

The length, width, and aspect ratios for the combined fibers and fiber clusters are illustrated in Figs. 1–3. Figures 1A–D illustrate fiber length characteristics. Those fibers or fiber clusters having a diameter greater than 0.6 μm are shown by the crosshatched portions in these figures. This cutoff was chosen because fibers

Allentown (cont.)
Honeckman, Charles, DO
Klass, Athanasius C., MD
Kostanik, Frances V., MD
Little, Brian W., MD, PhD
Scott, Ralph H., MD

Allison Park
Cruz, Pedro A., MD
Dekker, Andrew, MD
Edwards, Rosemary, MD
Specht, Charles S., MD

Altoona
Jourdain, Luis M., MD
Kirsch, William J., MD
Snell, Eugene M., MD
Stanfield, Barbara L., MD

Alum Bank
Sharpe, Earl S., MD

Ambler
Hagarty, John J., MD

Annville
Carmany, Thomas S., MD

Ardmore
Carroll, Deborah J., MD
Smith, M. Sue, MD
Wasik, Mariusz A., MD

Aspinwall
Finkelstein, Sydney D., MD

Audubon
Nikitas, Donald A., MD

Bala Cynwyd
Sun, Yongling, MD
Dotson, David James, MD
Dotson, Lynn H., MD

Bala-Cynwyd
Forest, Jean L., MD
Meyers, Karl R., MD
Uri, Antonia K., MD
Yarow, Noemi Susana, MD

Beaver
Saglio, Corrado M., MD
Marcus, Gary J., MD
Min, Tae C., MD
Reitz, John O., MD

Berwick
Hunter, Robert Gray, MD

Berwyn
Schramm, Harry A., MD

Bethel Park
Pradhan, Sulochana L., MD
Schubert, Eric D., MD

Bethlehem
Benz, Edward J., MD
Chiadis, James M., MD
Fisher, Joseph W., Jr., MD
Gonzalez, Rafael O., MD
Isaac, Leon A., MD
Longo, Santo, MD
Luft, Ronald D., MD
Lukaszczyk, Thomas A., MD
Mihalakis, Isidore, MD
Oliver, Jorge M., MD

Bloomsburg
Donon, R. Patrick, MD
Martin, John C., MD
Tilman, William E., MD

Blue Bell
Lieberman, Jill Mazzei, MD

Boothwyn
Campbell, William N., MD

Bowmansville
Lane, C. Darrell, MD

Braddock
Castro, Augusto D., MD
Kritcher, Charles, MD

Bristol
Song, Sang Won, MD

Brookhaven
Wilson, Roma A., MD

Brookville
Haverty, Gary F., DO

Brossmall
Daum, Gary S., MD

Bryn Mawr
Hausman, David H., MD
Hemert, Samia R., MD
Kasbegelan, Albert A., MD, PhD
Ledoulx, Charles T., MD
Miguel, Nemesio L., Jr., MD
Pietra, Giuseppe G., MD
Strumia, Paul V., MD
Williams, James R., MD

Butler
Kim, Yang K., MD
Moon, Sanggu, MD
Pirrello, Anthony M., Jr., MD

Camp Hill
Al-Annouf, Nabil, MD
Jenkins, Rosemary, MD
Maas, Anthony E., MD
Meniglia, Rosario, MD
Potok, Julian W., DO

Campbell
Bhattacharji, Susanta K., MD

Canonsburg
Syed, Tasneem A.Z., MD

Carlisle
Chang, Duck-Kyu, MD
Crist, Henry S., MD
Smith, James M., MD

Cedar Run
Corson, Margaret L., MD

Chadds Ford
Meier, Frederick A., MD

Chalfont
Aegerter, Ernest E., MD

Chalk Hill
Ayres, William W., MD

Chambersburg
Flanagan, Margaret M., MD
Hoffman, Howard L., MD
Ramirez, Constancio A., MD

Chester
Balin, Arthur K., MD, PhD
Lewis, James, DO
Spector, Harvey S., MD

Clarion
Puckett, James L., DO

Clarks Summit
O'Connor, James J., Jr., MD
Ross, Gary W., MD

Clearfield
Kennard, John F., MD
Reed, Michael F., MD

Coatesville
Casas, Angelina W., MD

Columbia
Sator, Susan M., MD

Conshohocken
Foldes, Julius, MD

Cooperstown
Lesko, Linda S., MD

Courtdale
Hudock, George E., Jr., MD

Dallas
Kashi, C. Warren, Jr., MD
Michalak, William A., MD
Wilde, William L., MD

Danville
Brett, David A., DO
Cera, Peter J., Jr., MD
Fevino, C. James, MD
Garbes, Archimedes D., MD
Hamati, Husam F., MD
Moran, John J., MD
Pascucci, Mary F., DO
Pierce, Richard N.
Rigotti, Richard M., MD
Schuerch, Conrad, MD
Wadih, George E., MD

Darby
Zotwaka, Maleka, MD

Devon
Moreno, Carlos, MD
Szymanski, Mark S., MD

Doylestown
Hawksley, Vaughn C., MD

Dresher
Schwartz, Heinz G., MD

Drexel Hill
Choi, John K., MD, PhD
Matthews, Lawrence M., Jr., MD
Monte, Steven A., MD

Du Bois
Costa, Jose M.L., MD

DuBois
Suslow, Gregory R., MD

East Stroudsburg
Cerra, Carmine J., MD
Gruszka, Frances A., MD
Tinsley, John P., MD

Easton
Altman, Arthur A., MD
Barnett, Charles P., MD
Brown, John M., MD
Dorman, Sandy A., MD
Fonseca, Carlos A., MD
Gaulin, J. Claude, MD
Harada, William A., MD
Salcedo, Wilfredo G., MD

Elkwood City
Williams, Karl E., MD

Elysburg
Reuter, Robert K., DO

Emmaus
Matalewicz, Theodore J., MD

Ephrata
Cota, Peter Christopher, MD

Erie
Eisenberg, Richard B., MD
Franklin, Norman, MD
German, Antonio L., MD
Jargess, Kenneth H., MD
Khera, Ritu, MD
Kiawon, David L., MD
Lara, Henry R.C., MD
Reitz, Mary E., MD
Rezwadewski, Jack V., MD
Ziegler, Thomas R., MD

Export
Talamo, Thomas S., MD

Farrell
Wilson, Steven Henry, MD

Fort Washington
Catalano, Edison, MD
Smith, Roberta Elaine, DO

Franklin
Davis, Bridgett K., MD
Sheppard, John W., MD
Suk, Jin H., MD

Gettysburg
Crippen, John W., MD
Mondelair Magdalena O., MD

Registrars for LAP printed in bold type.

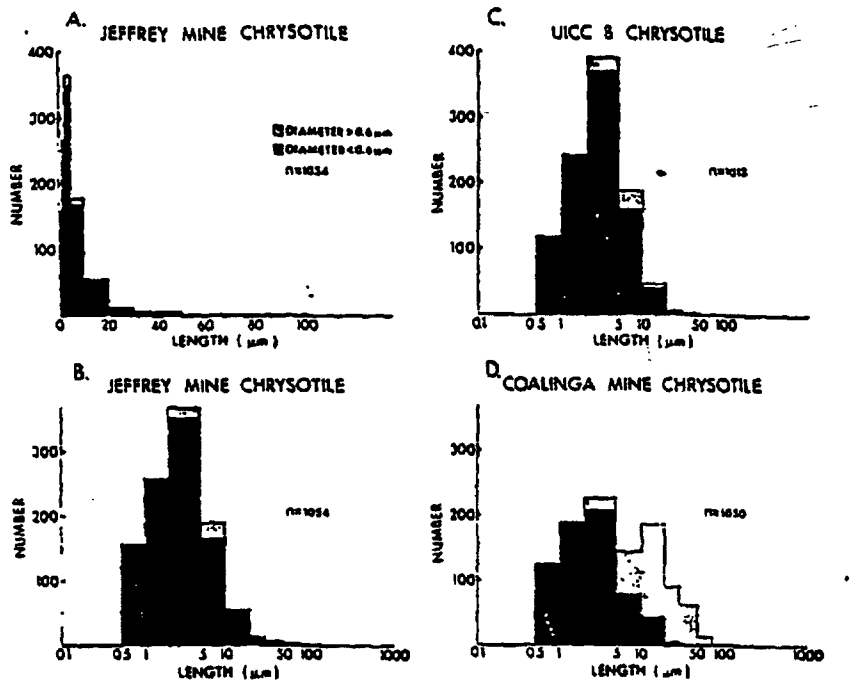

FIG. 1. Frequency distribution of length for combined fibers and fiber clusters in each of the aerosolized chrysotile preparations. (A) Jeffrey Mine chrysotile plotted on a linear scale for length. (B–D) Chrysotile preparations plotted on a log scale for length.

fiber clusters, each as a separate category. The fibers and fiber clusters are expressed as numbers counted per length interval, the percentage of the total found in each length interval, the cumulative percentage, and the mean diameter of fibers or fiber clusters in each length interval. The fibers and fiber clusters for each preparation are also expressed in terms of their respective aspect ratios in Tables 3–5.

A comparison of Tables 3–5 demonstrates that the mean diameter of fibers or fiber clusters for any given length interval is similar for the Jeffrey and UICC B preparations. There was no fiber or fiber cluster measured in either the Jeffrey or UICC B preparation that exceeded 2 μm in diameter. The aspect ratio increased for both fibers and fiber clusters with increasing length in the Jeffrey and UICC B preparations. The Coalinga preparation has a similar mean fiber and fiber cluster diameter below the 5-μm length interval. However, for fibers and fiber clusters longer than 5 μm in length, the mean diameter in the Coalinga preparation is significantly greater for both fibers and fiber clusters compared to the Jeffrey and UICC B preparations. In general, as the fiber or fiber cluster length increased in the Coalinga preparation, the width also increased. The presence of "thick" fibers

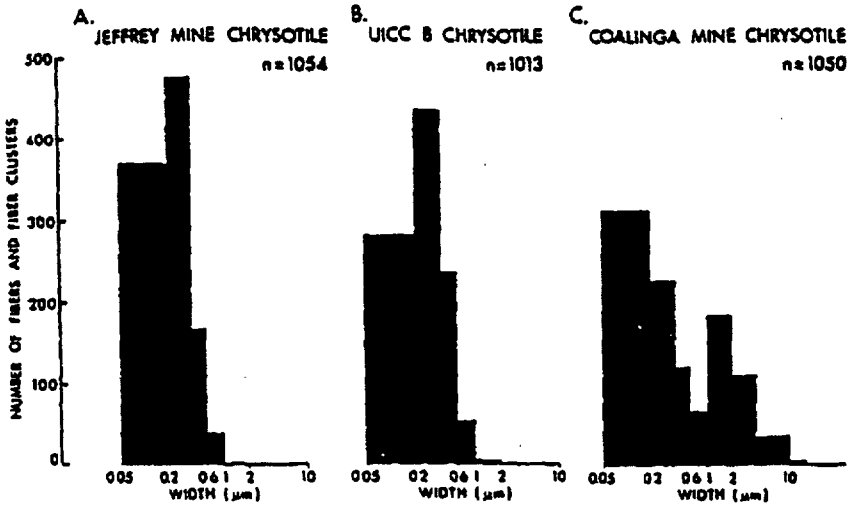

FIG. 2. Frequency distribution of log width for combined fibers and fiber clusters in each aerosolized chrysotile preparation.

and "thick" fiber clusters in the Coalinga preparation is reflected in the smaller aspect ratios which in any length interval seldom exceeded 60:1.

The results of fiber counting by optical microscopy cannot be expected to correlate closely with those obtained by scanning electron microscopy. The criteria for fiber counting by optical microscopy includes all fibers less than 3 μm

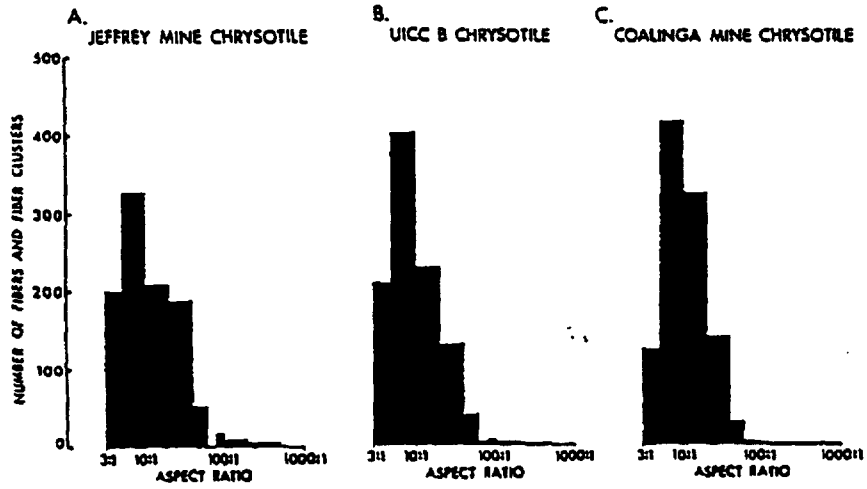

FIG. 3. Frequency distribution of aspect ratio for combined fibers and fiber clusters in each aerosolized chrysotile preparation. (n = 1054, 1013, and 1050, respectively.)

TABLE 3
PARTICLE SIZE DISTRIBUTION OF JEFFREY MINE CHRYSOTILE IN THE AEROSOLIZED STATE

	Length interval (μm)								
	0–0.99	1–1.99	2–4.99	5–9.99	10–19.9	20–29.9	30–49.9	50–99.9	>100
Fiber, n = 767									
Number	137	205	250	110	42	13	5	4	1
Percentage of total	17.9	26.7	32.6	14.3	5.5	1.7	0.7	0.6	0.1
Cumulative percentage	17.9	44.6	77.2	91.5	97.0	98.7	99.3	99.9	100
Mean diameter	0.20	0.22	0.28	0.29	0.31	0.48	0.30	0.50	0.50
Percentage of fibers by aspect ratio									
3:1–4.99:1	100	3	2	0	0	0	0	0	0
5:1–9.99:1	0	97	5	0	0	0	0	0	0
10:1–19.9:1	0	0	56	10	0	0	0	0	0
20:1–39.9:1	0	0	37	68	17	8	0	0	0
40:1–59.9:1	0	0	0	22	67	0	20	0	0
60:1–79.9:1	0	0	0	0	0	0	0	0	0
80:1–99.9:1	0	0	0	0	17	69	20	0	0
100:1–199:1	0	0	0	0	0	23	60	50	0
200:1–499:1	0	0	0	0	0	0	0	50	100
>500:1	0	0	0	0	0	0	0	0	0
Fiber cluster, n = 287									
Number	21	62	117	68	14	1	2	2	0
Percentage of total	7.3	21.6	40.8	23.7	4.9	0.3	0.7	0.6	0.0
Cumulative percentage	7.3	28.9	69.7	93.4	98.3	98.6	99.3	100	100
Mean diameter	0.30	0.34	0.44	0.48	0.46	0.8	0.4	0.6	—
Percentage of fiber clusters by aspect ratio									
3:1–4.99:1	100	100	11	1	0	0	0	0	0
5:1–9.99:1	0	0	89	15	0	0	0	0	0
10:1–19.9:1	0	0	0	84	14	0	0	0	0
20:1–39.9:1	0	0	0	0	86	100	0	0	0
40:1–59.9:1	0	0	0	0	0	0	0	0	0
60:1–79.9:1	0	0	0	0	0	0	0	0	0
80:1–99.9:1	0	0	0	0	0	0	0	0	0
100:1–199:1	0	0	0	0	0	0	100	100	0
200:1–499:1	0	0	0	0	0	0	0	0	0
>500:1	0	0	0	0	0	0	0	0	0
Nonfibrous particle, n = 174									
Number	105	47	22	0	0	0	0	0	0

TABLE 4
PARTICLE SIZE DISTRIBUTION OF UICC B CHRYSOTILE IN THE AEROSOLIZED STATE

Length interval (μm)

TABLE 4

PARTICLE SIZE DISTRIBUTION OF UICC B CHRYSOTILE IN THE AEROSOLIZED STATE

	Length interval (μm)								
	0–0.99	1–1.99	2–4.99	5–9.99	10–19.9	20–29.9	30–49.9	50–99.9	>100
Fiber, n = 610									
Number	104	181	211	75	26	8	3	1	1
Percentage of total	17.0	29.7	34.6	12.3	4.3	1.3	0.5	0.2	0.2
Cumulative percentage	17.0	46.7	81.3	93.6	97.8	99.1	99.6	99.8	100
Mean diameter	0.19	0.23	0.30	0.34	0.34	0.38	0.36	0.30	0.80
Percentage of fibers by aspect ratio									
3:1–4.99:1	100	2	1	0	0	0	0	0	0
5:1–9.99:1	0	98	14	3	0	0	0	0	0
10:1–19.9:1	0	0	53	25	0	0	0	0	0
20:1–39.9:1	0	0	31	49	23	0	0	0	0
40:1–59.9:1	0	0	0	23	73	38	0	0	0
60:1–79.9:1	0	0	0	0	0	0	0	0	0
80:1–99.9:1	0	0	0	0	4	63	33	0	0
100:1–199:1	0	0	0	0	0	0	67	0	100
200:1–499:1	0	0	0	0	0	0	0	100	0
>500:1	0	0	0	0	0	0	0	0	0
Fiber cluster, n = 403									
Number	16	62	181	115	23	5	1	0	0
Percentage of total	4.0	15.4	44.9	28.5	5.7	1.2	0.2	0.0	0.0
Cumulative percentage	4.0	19.4	64.3	92.8	98.5	99.8	100	100	100
Mean diameter	0.30	0.30	0.44	0.51	0.41	0.67	0.40	—	—
Percentage of fiber clusters by aspect ratio									
3:1–4.99:1	100	100	9	1	0	0	0	0	0
5:1–9.99:1	0	0	91	21	0	0	0	0	0
10:1–19.9:1	0	0	0	78	26	0	0	0	0
20:1–39.9:1	0	0	0	0	74	60	0	0	0
40:1–59.9:1	0	0	0	0	0	0	0	0	0
60:1–79.9:1	0	0	0	0	0	40	0	0	0
80:1–99.9:1	0	0	0	0	0	0	0	0	0
100:1–199:1	0	0	0	0	0	0	100	0	0
200:1–499:1	0	0	0	0	0	0	0	0	0
>500:1	0	0	0	0	0	0	0	0	0
Nonfibrous particle, n = 138									
Number	105	27	6	0	0	0	0	0	0

TABLE 5
PARTICLE SIZE DISTRIBUTION OF COALINGA MINE CHRYSOTILE IN THE AEROSOLIZED STATE

	Length interval (μm)									
	0–.99	1–1.99	2–2.99	3–4.99	5–9.99	10–19.9	20–29.9	30–49.9	50–99.9	>100
Fiber, n = 818										
Number	125	188	112	93	110	117	48	20	5	0
Percentage of total	15.3	23.0	13.7	11.4	13.4	14.3	5.9	2.4	0.6	0.0
Cumulative percentage	15.3	38.3	52.0	63.3	76.8	91.1	96.9	99.4	100	100
Mean diameter	0.15	0.20	0.26	0.39	0.71	1.08	2.15	2.36	2.92	—
Percentage of fibers by aspect ratio										
3:1–4.99:1	52	3	0	5	11	3	6	5	0	—
5:1–9.99:1	48	75	60	27	13	25	19	5	0	—
10:1–19.9:1	0	22	20	48	45	26	40	35	40	—
20:1–39.9:1	0	0	11	16	25	33	23	45	20	—
40:1–59.9:1	0	0	0	3	5	12	10	5	20	—
60:1–79.9:1	0	0	0	0	1	0	0	0	0	—
80:1–99.9:1	0	0	0	0	0	1	2	5	20	—
100:1–199:1	0	0	0	0	0	0	0	0	0	—
200:1–499:1	0	0	0	0	0	0	0	0	0	—
>500:1	0	0	0	0	0	0	0	0	0	—
Fiber cluster, n = 232										
Number	1	3	4	19	37	72	44	42	9	1
Percentage of total	0.4	1.3	1.7	8.2	15.9	31.0	19.0	18.1	3.8	0.4
Cumulative percentage	0.4	1.7	3.4	11.6	27.6	58.6	77.6	95.7	99.6	100
Mean diameter	0.30	0.30	0.40	0.59	0.95	1.79	2.42	3.32	6.17	5.5
Percent of fiber clusters by aspect ratio										
3:1–4.99:1	100	100	100	16	22	13	2	2	11	0
5:1–9.99:1	0	0	0	26	22	42	25	26	11	0
10:1–19.9:1	0	0	0	58	56	31	50	38	67	0
20:1–39.9:1	0	0	0	0	0	8	23	33	11	100
40:1–59.9:1	0	0	0	0	0	7	0	0	0	0
60:1–79.9:1	0	0	0	0	0	0	0	0	0	0
80:1–99.9:1	0	0	0	0	0	0	0	0	0	0
100:1–199:1	0	0	0	0	0	0	0	0	0	0
200:1–499:1	0	0	0	0	0	0	0	0	0	0
>500:1	0	0	0	0	0	0	0	0	0	0
Nonfibrous particle, n = 224										
Number	143	46	16	12	7	0	0	0	0	0

in diameter and greater than 5 μm in length. The criteria for fiber–fiber cluster counting by scanning electron microscopy includes fibers of all lengths and diameters so long as they possess at least a 3 to 1 aspect ratio. By these standards, 75% of the fiber–fiber clusters in the aerosolized Jeffrey and UICC B preparations counted by scanning electron microscopy would not have been counted by optical microscopy. For the aerosolized Coalinga preparation, 60% of the fiber–fiber clusters would not have been counted by optical microscopy (10% of the total fiber–fiber cluster number exceeded the 3-μm-diameter limit and 50% of the total fiber–fiber cluster number was less than 5 μm in length).

Nonfibrous Particle Analysis

The elements detected in the 50 randomly selected nonfibrous particles for each chrysotile preparation are shown in Table 6. One half of the nonfibrous particles analyzed in the Jeffrey and UICC B preparation samples demonstrated the presence of magnesium and silicon only. The magnesium-to-silicon ratio for fibers and fiber clusters analyzed in all three preparations ranged from 0.62 to 1.34. Although not a means of identification, particles with a Mg-to-Si ratio in this range are likely to represent fragments of chrysotile. The percentage of particles possessing the same Mg-to-Si ratio as chrysotile is 40% in the Jeffrey Mine preparation, 52% in the UICC B reference sample, and 20% in the Coalinga Mine preparation. The particles with low magnesium-to-silicon ratios of 0.30 to 0.40, found only in the Jeffrey Mine aerosolized sample, may represent talc particles.

DISCUSSION

Two of the chrysotile preparations designed for experimental research and characterized in this study have not been previously studied in the aerosolized state. UICC B chrysotile has been studied extensively by both Timbrell (1970a) and Beckett (1973) in the aerosolized state. To fit the Coalinga Mine and Jeffrey Mine samples into the spectrum of chrysotile preparations available for experimental inhalation research, UICC B chrysotile was used as a reference to link the present study to the work carried out by other investigators, in particular, the aerosolization studies of Timbrell and Beckett. Although differences exist in the manner of fiber collection from the dust chambers and in the preparation and analysis of samples, the studies of Timbrell and Beckett on UICC B are comparable to the present study (Table 7). By light microscopy we found a greater proportion of the fibers falling into longer fiber length intervals than did the studies of Beckett and Timbrell. This difference may reflect differences in fiber preparation and/or counting techniques. By electron microscopy, the results of this study for UICC B chrysotile are nearly identical to those of Timbrell for the distribution of fiber lengths. Beckett (1973) used SEM to determine fiber length and evaluated only fibers longer than 5 μm. When our present results were recalculated and expressed in a similar fashion (Table 7), we found a similar pattern, but proportionally fewer long fibers than did Beckett. Some of these differences may be due to differences in the manner in which the aerosols were generated and the samples collected.

The Jeffrey Mine and Coalinga Mine chrysotiles have been elegantly analyzed

TABLE 6
ELEMENTAL COMPOSITION OF THE NONFIBROUS PARTICLES IN EACH CHRYSOTILE PREPARATION[a]

Elements detected	Percentage occurrence		
	Jeffrey	UICC B	Coalinga
NaMgAlSiCa	2	—	—
NaAl	—	—	6
NaAlSiKCaFe	2	—	—
Mg	2	2	4
MgAl	4	—	—
MgAlSi	14	30	10
MgAlSiKFe	2	—	—
MgAlSiCaFe	—	2	—
MgAlSiCrFe	—	—	2
MgAlSiFe	6	—	2
MgSi ratio	50	54	20
Mg to Si = 0.3–0.4	(6)	(—)	(—)
Mg to Si = 0.6–1.1	(40)	(52)	(20)
Mg to Si = 2.0–10	(4)	(2)	(—)
MgSiCa	—	2	—
MgSiFe	—	—	6
MgK	—	2	—
Al	2	4	14
AlSi	6	—	4
AlSiCa	2	—	—
AlSiK	—	—	2
AlSiCr	—	—	4
AlSiFe	—	—	2
AlK	2	—	—
AlKCa	—	—	2
AlCr	2	—	14[b]
SiCr	—	—	2
CaCr	—	—	2
Cr	—	—	4[b]
Fe	2	—	—
None	2	4	—

[a] 50 random nonfibrous particles were analyzed for each chrysotile preparation.
[b] May represent a contaminant from the rotary blade used to aerosolize the preparation.

by others using the sample preparation technique of fiber dispersion in a liquid medium (Wylie, 1979; Campbell *et al.*, 1980; Siegrist and Wylie, 1980). This type of analysis is satisfactory for studies in which the preparations are to be ingested or injected in suspension. However, these studies do not provide an adequate characterization of the asbestos preparations for inhalation studies since the process of aerosolization is generally less efficient in fiber dispersion. A case in point is the Coalinga Mine chrysotile preparation. This asbestos preparation was characterized by Campbell *et al.* (1980) using fiber samples which were dispersed in a liquid medium by ultrasonification for 10 min. The samples were analyzed by transmission electron microscopy. They found that 2.1% of the total chrysotile

TABLE 7

FIBER LENGTH DISTRIBUTION—OPTICAL MICROSCOPY

Preparation	Dispersion Method	Study	Percentage longer than stated length (μm) 4	5	10	20	40
UICC B	Aerosol	Present		100	68.2	40.5	12.6
UICC B	Aerosol	Beckett[a]		100	32	11	5
UICC B	Aerosol	Timbrell[b]	100		22.3	11	1.7
UICC B	Alcohol	Timbrell[b]		100	46.9	21.5	7.0
UICC B	Celloidin	Timbrell[b]		100	53.6	16.9	3.8

FIBER LENGTH DISTRIBUTION—ELECTRON MICROSCOPY

Asbestos-dispersion method	Instrument	Study	Percentage longer than stated length (μm) 0.2	1	2	5	10	20
UICC B aerosol	TEM	Timbrell[b]	100	73.6	54.4	27.0	9.6	3.2
UICC B aerosol	SEM	Present	100	88.2	64.2	25.5	6.7	1.9
JEFFREY aerosol	SEM	Present	100	85.0	50.7	24.9	8.0	2.7
			5	10	20	30	50	60
UICC B aerosol	SEM	Beckett[a]	100	45	12	6		1
UICC B aerosol	SEM	Present	100	26.4	7.4	2.3	0.8	
JEFFREY aerosol	SEM	Present	100	32.1	10.8	5.5	2.7	

[a] From Figure 2a in Beckett, 1973.

[b] Modified from Tables 6, 7, or 8 in Timbrell, 1970a.

particles analyzed were greater than 10 μm in length and had a mean diameter of 0.42 μm. In the present study using aerosolized samples drawn directly upon Nucleopore filters and subsequently gold coated, it was found that 34.0% of the combined fibers and fiber clusters or 28.1% of all particles (fibers, fiber clusters, and nonfibrous particles) in the Coalinga preparation were greater than 10 μm in length and had a mean diameter of 2.92 μm. In a subsequent study by Siegrist and Wylie (1980), the Coalinga preparation (referred to as short-range chrysotile) was characterized for particle size distribution by both transmission and scanning electron microscopy. The samples were prepared by hand swirling in distilled water and dishwashing liquid (for SEM analysis) and by ultrasonification in water for 10 min (for TEM analysis). The results demonstrated that by both SEM and TEM more than 90–95% of the particles were less than 10 μm in length and more than 95% of the particles were less than 1 μm in diameter. In our study using aerosolized samples 71.9% of the total number of particles were less than 10 μm in length and 68% of the total number of particles were less than 1 μm in diameter.

The differences between these two methods of sample preparation and analysis

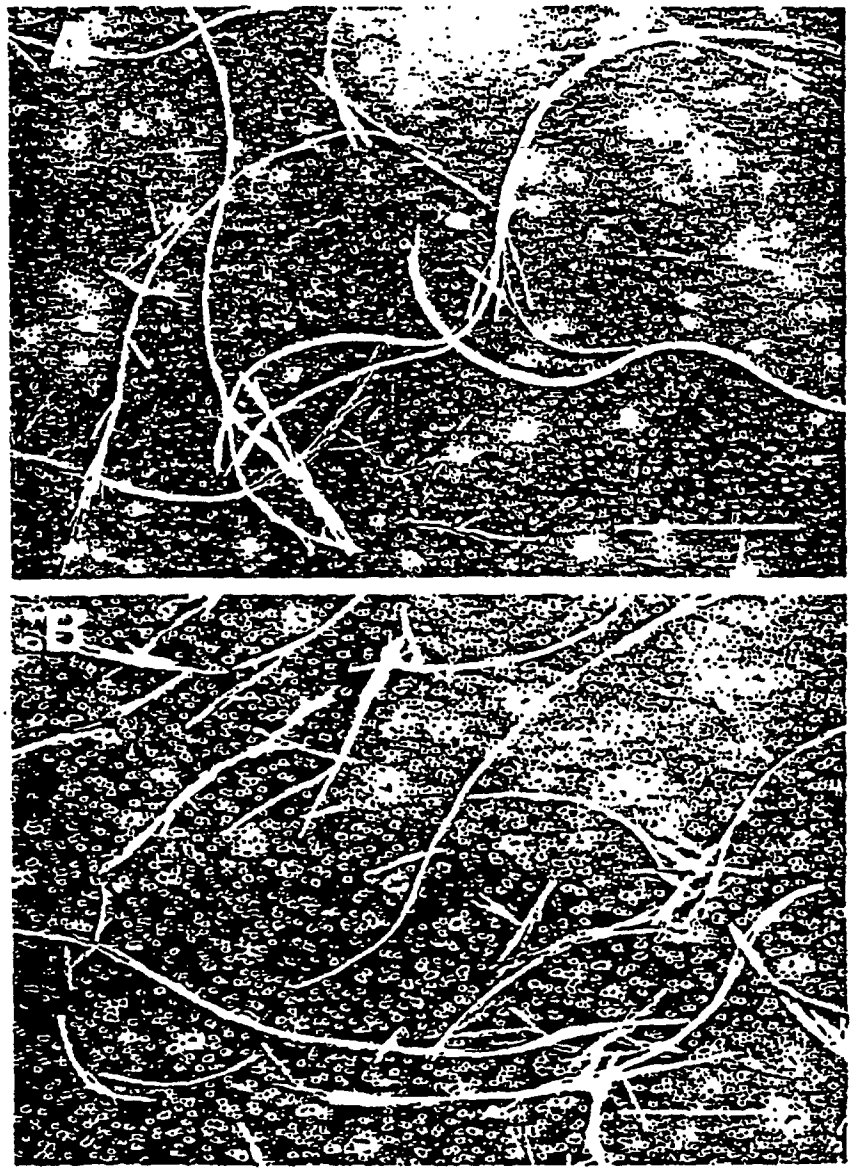

FIG. 4. (A) Aerosolized Jeffrey Mine chrysotile collected on a 0.2-μm Nucleopore filter and gold coated. Bar = 10 μm. (B) Aerosolized UICC B chrysotile. Bar = 10 μm. (C) Aerosolized Coalinga Mine chrysotile. Bar = 10 μm. Examples of a fiber (short arrow) and a fiber cluster (long arrow) are indicated in this micrograph. (D) Higher magnification of an aerosolized Coalinga Mine chrysotile fiber cluster demonstrating the fibrillar subunit composition. Bar = 1 μm.

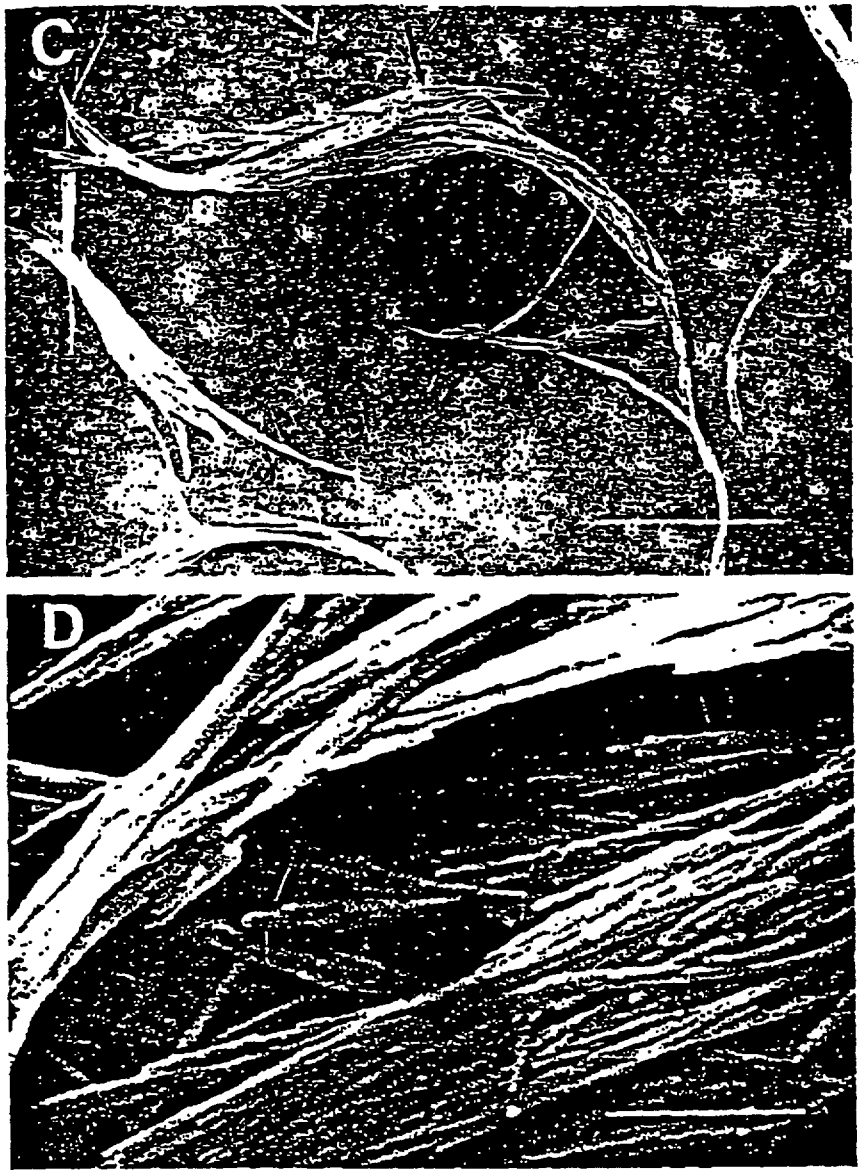

FIG. 4—*Continued.*

suggest that either ultrasonification breaks down fiber bundles into smaller bundles and fibrils or that fibers tend to cluster in the aerosolized state. Other investigators have shown that ultrasonification can result in the breakdown of chrysotile into smaller fibers (Spurny *et al.*, 1980). In addition, clustering of fibers caused by the aerosolization does not seem likely since the Jeffrey and UICC B chrysotile preparations collected in the same manner as the Coalinga chrysotile have distinctly different particle size distributions from that seen in the aerosolized Coalinga preparation. The illustration of each fiber preparation on the Nucleopore filters (Fig. 4) also show a distinct fiber morphology for the Coalinga chrysotile compared to the Jeffrey and UICC B chrysotile.

Some of the differences between each of the chrysotile preparations were also reflected by the gravimetric measurements taken of each chrysotile aerosol in the exposure chambers. Comparisons made between each chrysotile preparation of the ratio of respirable mass concentration to total mass concentration demonstrate a distinct difference between the Coalinga chrysotile and the other two preparations of Jeffrey and UICC B chrysotile. It would appear that compared to the Jeffrey and UICC B fibers the Coalinga preparation contains a greater proportion of particles which are captured in the elutriator system of the Casella sampler and in the precutter stages of the Cascade impactor. These fibers and particles are captured because of their greater mass.

Using scanning electron microscopy we found that the range of diameters of fibers and fiber clusters was greater for the Coalinga preparation than for the Jeffrey and UICC B preparations in the aerosolized state. The trend of increasing diameter with increasing length was found only in the Coalinga preparation. Fibers and fiber clusters greater than 10 μm in length with diameters exceeding 2 μm were present in the aerosolized Coalinga preparation while in the Jeffrey and UICC B preparations no fiber or fiber cluster of any length exceeded 2 μm in diameter.

The characterization of chrysotile in the aerosolized state in terms of fiber length and fiber diameter is paramount in understanding the nature of any preparation used for inhalation studies. Although the physical characteristics of a fiber may not be the only factor in causing lung injury, fiber diameter and, to a lesser degree, fiber length play key roles in the potential for a fiber to reach the alveolar regions of the lung where injury as a result of asbestos inhalation appears to be more severe.

The potential for each of these chrysotile preparations to cause lung injury by inhalation can be best assessed by a review of what is known about the physical characteristics a fiber must possess in order to reach the alveolar portions of the lung. Spherical particles below a certain diameter (approximately 3–4 μm) can reach the alveolar airspaces of the lung (Lippman and Albert, 1969). Larger particles are eliminated by deposition in the upper airways primarily through sedimentation and impaction as a result of their greater mass. Timbrell, Harris and Fraser have examined the more complex aerodynamic properties of fibers by comparing the deposition pattern of fibers to those of artificial spheres (Timbrell, 1965). In general they found that fibers have a similar deposition pattern to that of spheres of three to four times greater diameter. This would suggest that fibers

much greater than 1 μm in diameter would not be likely to reach the alveolar airspaces although the curvature of the fiber should also be taken into consideration (Timbrell, 1970b). Additional studies have shown that an equivalent fiber diameter of 0.5 to 2.0 μm results in deposition within respiratory bronchioles and proximal alveolar ducts, while fibers of smaller equivalent diameters result in maximal deposition in the more distal portions of the lung (Harris and Fraser, 1976). Pooley and Clark (1979) measured the diameter of chrysotile fibers recovered from lung specimens after tissue digestion. They found that only 0.06% of the fibers had a diameter exceeding 0.5 μm. Although it is possible that these chrysotile fibers have fragmented longitudinally while *in vivo* (Suzuki and Churg, 1969), these results suggest that few fibers with a diameter greater than 0.5 μm reach the alveolar regions of the lung. If this limit in fiber diameter is true for alveolar lung deposition and the length of the fiber plays the major role in fibrogenesis and cell injury, the potential for the three asbestos preparations reported in this study to cause lung injury by inhalation will be different. Only the Jeffrey and UICC B chrysotile preparations have fibers which are less than 0.6 μm in diameter when length is greater than 30 μm. The Coalinga Mine chrysotile does not possess this property; instead, with increasing fiber length, fiber diameter also increases. Most fibers longer than 20 μm in length in the aerosolized Coalinga preparations are also greater than 0.6 μm in diameter. Thus, in an inhalation study, more fibers of greater length distribution would be deposited in the alveolar region of the lungs for the Jeffrey and UICC B chrysotile preparations than for the Coalinga chrysotile preparation.

At this point one may ask the question, what constitutes a short-range fiber preparation? We have seen that the Coalinga preparation originally thought to be a short-fiber preparation contains numerous long fibers in the aerosolized state. The answer to this question should be based on the potential of a fiber to reach the alveolar regions of lung. In other words, is the fiber respirable? The work of investigators who have studied the aerodynamic properties of fibers (Timbrell, 1965, 1973) and the physical dimensions of respired fibers (Pooley and Clark, 1979), would suggest that the Coalinga chrysotile in the aerosolized state represents a short-fiber preparation since only fibers less than 30 μm in length are likely to be respirable. In contrast, the Jeffrey and UICC B chrysotile preparations contain fibers greater than 30 μm in length possessing a diameter which would permit penetration of the fiber into the alveolar regions of the lung. Based on these observations, the Coalinga Mine chrysotile preparation can be considered to represent a "shorter" fiber preparation in the aerosolized state.

In summary, the three chrysotile preparations characterized in the aerosolized state in this study demonstrated distinct properties. Gravimetric measurements of each chrysotile preparation revealed that both the Jeffrey and UICC B preparations have a significantly greater portion of the total chamber dust concentration which is respirable compared to the Coalinga preparation. By light microscopy it was found that for fibers greater than 5 μm in length, the Jeffrey preparation in the aerosolized state has a significantly greater fraction of fibers exceeding 40 μm in length than does UICC B chrysotile or Coalinga chrysotile. Finally, by scanning electron microscopy it was found that the Jeffrey and UICC B preparations pos-

sess fibers and fiber clusters which cross a large length range (the longest measured was 150 μm), but none which exceeded 2 μm in diameter. The Coalinga preparation also possessed fibers and fiber clusters across a similar length range, but many exceeded 2 μm in diameter. In terms of respirability, the Jeffrey and UICC B aerosolized fibers constitute a mixed short-range and long-range fiber preparation, while the Coalinga aerosolized fibers represent a somewhat short-fiber preparation in which very long respirable fibers are not present. In applying the above fiber characterization studies to experimental research it is essential to remember that further manipulation of these chrysotile preparations by grinding (Langer *et al.*, 1978) or by fiber separation techniques may alter the fiber size distribution from that presented in this paper.

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Report 29-55

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MELLON INSTITUTE

Special Report

EXHIBIT

PIFX-23

11-28-79

The Fibrogenic Potential of Asbestos Proc----

via Intraperitoneal Injection in Guinea Pigs, Rats and Rabbits

and by the Intratracheal Route in the Rat

Chemicals Division, Union Carbide Corporation Industrial Fellowship 274-29

Summary

The results of intraperitoneal administration indicate that the asbestos products studied produced fibrotic lesions of the visceral organs in rats and guinea pigs regardless of fiber length. Of the three products, CMS-100 (refined fiber), produces the most severe reaction. JT-100 (standard fiber) and JM-3K (Johns Manville screened fiber) produced similar but less severe reactions. The results on rabbits were inconclusive.

Both JT-100 and CMS-100 produced specific lesions in rats by the intratracheal route but with JT-100 they were more severe. No lesions were found in the rats which received JM-3K fibers.

These results indicate that these three asbestos fibers may be fibrogenic by either route. No specific lesions were observed in the lungs of the JM-3K intratracheal rats, but only two rats survived to be examined.

Samples

The following samples were shipped to Mellon Institute at the request of Mr. Paul W. McDaniel who furnished the descriptive information.

Designation of Asbestos Product	Source	Quantity	Date Received	M. I. Sample Number
*CTS-100 Fiber (same as JT-100 Fiber) "Standard Fiber" containing impurities in the form of clay and magnetite. A short fiber.	Sterling Forest	8 oz.	8-14-63	26-220
*CMS-100 Fiber "Refined Fiber" with lesser impurities than JT-100. A short fiber.	Sterling Forest	8 oz.	8-14-63	26-219

4-4-5- Meyer. 4/4/82 RS

* The individual fibers for most part had lengths of 0.9 to 10 microns, but there were some as long as 30 microns and some that were less than 0.5 micron. The diameters of the fiber were generally 0.3 to 1.2 microns, but there were smaller sizes noted. The clay-magnetite particles were mostly 0.5 to 3.0 microns in diameter but [illegible]

Designation of Asbestos Product	Source	Quantity	Date Received	M. I. Sample Number
JM-3K Fiber. "Long Fiber" (Brushed through screen to reduce fiber length to 1 mm. or less before injecting.)	Manville, N.J.	8 oz.	8-26-63	26-227

Intraperitoneal Injections

Methods

The JM-3K asbestos was brushed through a 1 mm. screen to reduce its fiber length to < 1000 micra (1.0 mm.) in order to facilitate dosing. The CMS-100 and JT-100 asbestos samples were used as received. Each asbestos product was prepared as a 2.5% (W/V) suspension in 0.85% saline. All needles, syringes and suspensions were sterilized prior to use. The animals were divided into three groups--each consisting of eleven male albino guinea pigs (P), six male albino rats (R), and two male albino New Zealand rabbits (H). The guinea pigs and rabbits received intraperitoneal injections of 2 ml. (50 mg.) and the rats, 1 ml. (25 mg.) of the respective asbestos suspensions. Eleven guinea pigs and six rats received control intraperitoneal injections of saline. There were no rabbit controls. All injections were made in the lower right quadrant of the abdomen. Tables 29-92 through 29-95 contain the results on individual animals.

Results

CMS-100 produced the most severe reaction in the form of granulomas with giant cells in six of the seven guinea pigs examined micropathologically. Typical granulomas with giant cells were observed in three of four rats and the one rabbit.

JT-100 asbestos fiber produced a granulomatous reaction in all of the 8 guinea pigs as well as the one rat subjected to micropathological examination. The rabbits had incidental lesions unrelated to the asbestos fibers.

The JM-3K asbestos fibers produced granulomas with fibers visible in five of the five guinea pigs examined. One of two rats had granuloma formation which was visible on gross examination.

Intratracheal Injections

Methods

One per cent (W/V) suspensions of JT-100, CMS-100 JM-3K and a 0.5% suspension of CMS-100 were prepared in 0.85% saline as for the intraperitoneal injections.

Female albino rats weighing in excess of 200 grams were given 1 ml. of the respective asbestos suspensions by intratracheal injection. The rats were anesthetized with saturated vapor of anhydrous diethyl ether. The trachea was exposed by blunt dissection and the injection was made directly and the incision closed with a Michael Wound Clamp.

Eight rats were injected with 1 ml. of a 1% JT-100 suspension (10 mg. asbestos). Eight rats were injected with 1 ml. of a 1% JM-3K suspension (10 mg. asbestos). Because of the flocculent properties of CMS-100, only three rats were dosed with 1 ml. of a 1% suspension (10 mg. asbestos). Eight rats were injected with 1 ml. of a 1% CMS-100 suspension (5 mg. asbestos). Six rats injected with physiological saline served as controls. Tables 29-96 through 29-99 contain results on individual animals.

Results

Specific lesions were found in most of the rats given CMS-100 and JT-100 that consisted of marked granuloma formation with bluish foreign body material present (magnetite?). Marked bronchiolitis obliterans was noted in most rats, also with bluish foreign body material within the lesions. The JT-100 rats had a more severe fibrotic reaction to this foreign material. Multinucleate giant cells were a common feature with the granulomas of the JT-100 rats but were conspicuously absent in the lungs of the rats given CMS-100. Micropathology was performed on only two rats receiving JM-3K asbestos. Neither of these animals had lesions similar to JT-100 or CMS-100.

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Intraperitoneal Injections of Asbestos CMS-100

Dose of Asbestos	Animal Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
50 mg. (2 ml. of 2.5% suspension)	P. 96813	20 D	Peritonitis; asbestos scattered throughout abdominal cavity.	Not examined.
	P. 96803	75 D	Peritonitis; adhesions of intestines to liver.	Not examined.
	P. 96815	240 D	Autolyzed.	Not examined.
	P. 96806	270 D	No asbestos in abdominal cavity.	Not examined.
	P. 96802	286 S	Multiple small adhesions of the omentum to the mesentery.	Several small, well marked granulomas with giant cells on the serosal surface of colon.
	P. 96807	286 S	Adhesions of liver to stomach. Deposits scattered in the intestinal mesentery.	Liver--marked granulomatous reaction on the surface. Granulomas--serosal wall.
	P. 96810	286 S	Omentumal adhesions to liver with deposits in the omentum.	Liver--well marked granulomas. Marked chronic granulomas in the intestine and diaphragm.
	P. 96811	286 S	Intestinal adhesions to midline and seminal vessels.	Intestinal, liver, skeletal muscle--adhesions to each other. Areas of giant cell formation.
	P. 96812	286 S	Areas of fibrosis in most of left liver lobes.	Colon--large cystic masses filled with debris. Lined by columnar epithelium.
	P. 96814	286 S	Adhesions--liver to stomach and intestines.	Liver--fibrosis and granuloma formation. Foreign body granulomas on abdominal wall.
	P. 96821	286 S	Adhesions of abdominal wall to liver, stomach and omentum with deposits.	Granulomatous lesions of the stomach, liver and skeletal muscle.
	H. 96737	60 D	Numerous deposits of asbestos adherred to intestines.	Not examined.,
	H. 96574	270 S	Multiple adhesions, ventral colon and urinary bladder.	Multiple well encapsulated granulomas present with innumerable giant cells.

(Continued)

Table 29-9

(Continued)

ose of Asbestos	Animal Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
5 mg. One ml. of 2.5% suspension)	R. 4571	20 D	Congested lungs and liver; asbestos scattered throughout abdominal cavity.	Not examined.
	R. 4678	170 D	Asbestos deposits throughout abdominal area.	Not examined.
	R. 4629	258 S	Nodules of white material in mesentery, ventral and abdominal wall.	Scattered granulomas in intestine and skeletal muscle.
	R. 4631	258 S	Deposits in the mesentery and ligaments of the testicle.	Multiple granulomas with giant cells and asbestos deposits.
	R. 4634	258 S	Chronic abscess pneumonia.	Chronic bronchopneumonia.
	R. 4660	258 S	Scattered deposits in mesentery and wall of small intestines. Adhesion of testes to intestines.	Large granulomas, well encapsulated.

Table 29

Intraperitoneal Injections of Asbestos JT-100

Dose of Asbestos	Animal Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
50 mg. (2 ml. of 2.5% suspension)	P. 96644	11 D	Peritonitis, asbestos scattered throughout abdominal cavity.	Not examined.
	P. 96651	150 D	Entire gut adherred together. Liver adherred to ventral wall and diaphragm.	Not examined.
	P. 96849	185 D	Multiple visceral adhesions of the asbestos to the ventral abdominal wall.	Not examined.
	P. 96834	286 S	Gray deposits in the ventral abdominal wall.	Scattered foreign body granulomas with slightly elongated fibers of asbestos in them.
	P. 96835	286 S	Severe multiple adhesions with pus incorporated.	Typical granulomas.
	P. 96837	286 S	Multiple intestinal adhesions. Gray deposits in the abdominal wall.	Adherred masses with many granulomas on the serosal surface.
	P. 96839	286 S	Omental adhesions to the liver with tiny deposits in this mesentery.	Typical connective tissue granulomas.
	P. 96840	286 S	Spleen adherred to ventral wall, stomach to diaphragm.	Typical connective tissue granulomas.
	P. 96841	286 S	Multiple tiny deposits on left liver lobes and left abdominal wall.	Numerous granulomas with foreign bodies present in them.
	P. 96847	286 S	Omental adhesions to the ventral tips of the liver.	Typical granulomas with foreign bodies, giant cells, fibrosis present.
	P. 96848	286 S	Deposits on the intestinal mesentery and abdominal wall with omental adhesions.	Typical connective tissue granulas.
	H. 96729	270 S	Heart covered by chronic connective tissue.	Essentially normal.
	H. 96734	270 S	Multiple serosal deposits scattered primarily on the colon and ventral wall.	Essentially normal.

(Continued)

Table 29-55

(Continued)

Dose of Asbestos	Animal Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
25 mg. (One ml. of 2.5% suspension)	R. 4576	36 D	Cannibalized.	Not examined.
	R. 4536	160 D	Autolyzed.	Not examined.
	R. 4534	200 D	Peritonitis, pleuritis, some fibrosis in pleural cavity.	Not examined.
	R. 4617	242 D	Pneumonia. Asbestos particles scattered throughout entire abdominal cavity.	Not examined.
	R. 4596	258 S	Slight deposits in the mesentery and omentum.	Intestine--typical granulomas with foreign bodies.

Table 2

Intraperitoneal Injections of Asbestos JM-3K

Dose of Asbestos	Animal Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
50 mg. (2 ml. of 2.5% suspension)	P. 96829	18 D	Peritonitis, asbestos adherred to ventral wall and small intestine.	Not examined.
	P. 96833	43 D	Intestines adherred to ventral wall.	Not examined.
	P. 96830	64 D	Scattered adhesions to ventral abdominal wall.	Not examined.
	P. 96826	76 D	Peritonitis, liver and stomach adherred to ventral abdominal wall.	Not examined.
	P. 96828	78 D	Peritonitis, asbestos scattered throughout abdominal cavity.	Not examined.
	P. 96832	86 D	Intestines adherred to ventral abdominal wall. Abscessed at point of adhesion.	Not examined.
	P. 96822	286 S	Multiple adhesions of the liver to the stomach, omentum, and intestines.	Foreign bodies. Granuloma attached to intestinal wall. Some granulomas in liver.
	P. 96823	286 S	Deposits on right seminal vessel.	Seminal vessel--granulomatous reaction with foreign material in soft tissue and mesentery.
	P. 96824	286 S	Greenish deposits in the ventral abdominal wall and omentum with adhesions to liver.	Foreign body granulomas with foreign material present.
	P. 96827	286 S	Midline omental adhesions to greenish deposits.	Deposits of foreign material and large granulomas on muscle.
	P. 96831	286 S	Liver adherred to ventral abdominal wall.	Tip of liver infectious; abscess formation, granuloma formation.
	H. 96764	60 D	No asbestos in peritoneal cavity.	Not examined.
	H. 96697	270 S	Asbestos at site of injection.	Essentially normal.
5 mg. (One ml. of 2.5% suspension)	R. 4628	35 D	Pneumonia. Intestine adherred to the ventral wall.	Not examined.
	R. 4622	230 D	Pasturella Pneumonia. Intestines adherred to abdominal wall with each other.	Not examined.
	R. 4623	234 D	Autolyzed.	Not examined.
	R. 4615	241 D	Pasturella Pneumonia.	Not examined.
	R. 4578	258 S	Adhesions of the omentum and mesentery.	Essentially normal.
	R. 4583	258 S	Middle ear infection.	Essentially normal.

Table 29-95

Intraperitoneal Injections of Saline Control

Dose of Saline	Animal Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
2 ml.	P. 96857	88 D	Pneumonia.	Not examined.
	P. 96856	156 D	Pneumonia.	Not examined.
	P. 96853	170 D	Pneumonia.	Not examined.
	P. 96858	207 D	Pneumonia.	Not examined.
	P. 96852	286 S	Essentially normal.	Essentially normal.
	P. 96859	286 S	Essentially normal.	Scattered areas of interstitial pneumonia.
	P. 96860	286 S	Emaciated. Liver massively reduced in size.	Liver necrosed and abscessed
	P. 96861	286 S	Essentially normal.	Essentially normal.
	P. 96862	286 S	Essentially normal.	Essentially normal
	P. 96863	286 S	Chronic nephritis in both kidneys.	Chronic interstitial nephritis.
	P. 96864	286 S	Essentially normal.	Essentially normal.
One ml.	R. 4484	12 D	Pneumonia.	Not examined.
	R. 4483	258 S	Chronic abscessed pneumonia.	Massive abscessed pneumonia.
	R. 4531	258 S	Essentially normal.	Essentially normal.
	R. 4540	258 S	Essentially normal.	Essentially normal.
	R. 4542	258 S	Essentially normal.	Essentially normal.
	R. 4557	258 S	Essentially normal.	Essentially normal.

Table 29-96

Intratracheal Injections of Asbestos CMS-100 to Rats

Dose of Asbestos	Rat Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
10 mg. (One ml. of 1% suspension)	84636	30 S	Pneumonia.	Pneumonia, numerous granulomas scattered throughout lung.
	84645	90 S	Pneumonia.	Pneumonia, lesions of bronchiolitis obliterans in several bronchi and bronchioles.
	84702	180 S	Chronic pneumonia.	Scattered granulomas; lesions of bronchiolitis obliterans with foreign material and thrombotic-like areas.
5 mg. (One ml. of 0.5% suspension)	72531	12 D	Cannibalized.	Not examined.
	81427	30 S	Lung--appeared normal.	Multiple granulomas; bluish material present in all granulomas.
	81433	90 S	No lesions.	Scattered small granulomas. Some bronchiolitis obliterans also apparent.
	81440	90 S	Chronic pneumonia.	Pneumonia; scattered granulomas; bronchiolitis obliterans.
	72528	180 S	Not recorded.	Scattered small granulomas.
	77679	180 S	Lipid foci in lungs.	Bronchiolitis obliterans. Chronic granulomas sans giant cells.
	77680	180 S	Gray foci in lungs.	Numerous scattered granulomas.
	81600	180 S	Pneumonia.	Multiple granulomas scattered throughout lung. Bronchiolitis obliterans.

Table 2.

Intratracheal Injections of Asbestos JT-100 to Rats

Dose of Asbestos	Rat Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
10 mg. (One ml. of 1% suspension)	84629	60 D	Large white areas on lung; atelectasis of the right lung.	Bronchiectasis. Areas of acute bronchopneumonia.
	84672	123 D	Pneumonia.	Not examined.
	84669	151 D	Pasturella Pneumonia.	Not examined.
	84633	30 S	Chronic pneumonia.	Multiple granulomas and bronchiolitis obliterans.
	84659	30 S	Chronic pneumonia.	Pneumonia; granulomas, large lesions of bronchiolitis obliterans.
	84674	90 S	Pneumonia.	Numerous granulomas, well marked bronchiolitis obliterans.
	84676	90 S	Chronic abscessed pneumonia.	Scattered granulomas.
	84631	180 S	Not recorded.	Granulomas, some bronchiolitis obliterans.

Table 29-98

Intratracheal Injections of Asbestos JM-3K to Rats

Dose of Asbestos	Rat Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
10 mg. (One ml. of 1% suspension)	84695	10 D	Entire lung was dark red.	Not examined.
	84635	13 D	Chronic pneumonia.	Not examined.
	81214	15 D	Pneumonia and emphysema.	Not examined.
	81171	50 D	Chronic pneumonia; bronchial lymph nodes, enlarged and gray.	Not examined.
	81161	30 S	Chronic pneumonia.	Chronic bronchopneumonia.
	84660	180 S	Chronic pneumonia and enlarged lymph nodes.	Chronic bronchopneumonia, a few scattered granulomas.

Table 29-99

Intratracheal Injections of Saline to Rats

Dose of Saline	Rat Number	Days to Death or Sacrifice	Gross Pathology	Micropathology
One ml.	78253	30 S	Essentially normal.	Essentially normal.
	78273	30 S	Essentially normal.	Essentially normal.
	78302	90 S	Essentially normal.	Essentially normal.
	78717	90 S	Azygous Obscessed.	Few areas of chronic pneumonia.
	84639	180 S	Essentially normal.	Essentially normal.
	84646	180 S	Essentially normal.	Essentially normal.
	84651	180 S	Essentially normal.	Essentially normal.
	84677	180 S	Essentially normal.	Essentially normal.

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Chemical Hygiene Fellowship
MELLON INSTITUTE
Carnegie-Mellon University

Calidria Asbestos-Resin Grade RG 244

Tracheal Insufflation of Rat Lungs with Interpretation of Pathology after 30, 60, 90 and 180 Days

Editor: C. P. Carpenter Contributors: D. L. Geary, Jr., E. R. Kinkead, R. C. Myers, D. J. Nachreiner
For: UNION CARBIDE CORPORATION, Chemicals and Plastics Operations Division

Sample

A 500-gram sample of Resin Grade RG 244 UCC Calidria Asbestos was received 11-30-70, from King City, California, pursuant to arrangements made by Paul McDaniel of the New York Office. The sample was identified by the Chemical Hygiene Fellowship #33-251.

Tracheal Insufflation

A 1% (W/V) suspension of the RG 244 sample was prepared in 0.85% saline. All needles, syringes and suspensions were sterilized prior to use. Either 1 ml or 0.5 ml amounts of the sterile 1% suspension were injected into the rat lung through the trachea, exposed by blunt dissection, after a midline cervical incision. Following injection of these 200 to 300 gram, male albino, Harlan Wistar rats the incisions were closed with Michael wound clamps until healing ensued.

A total of 13 rats were dosed with 1 ml and 15 with 0.5 ml of the 1% suspension while 11 control rats received 1 ml of sterile 0.85% NaCl. Three rats from each asbestos dosed group and 2 controls were killed for histopathologic examination of the lung after intervals of 30, 60 and 90 days which left groups of 4, 6 and 5 rats on the 1 ml, 0.5 ml asbestos and control for the 180 day sacrifice.

Summary of Microscopic Pathology Found 30, 60, 90 and 180 Days Following Tracheal Insufflation of Rats

The 30-day pathology was marked by the presence of granulation tissue with thickening of the structural elements of the lung (stroma) and the accumulation of giant cells often associated with foreign bodies.

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After 60 days one rat on the 0.5 ml dosage level had inflammation and ingrowth of connective tissue which blocked a terminal bronchus. Two of 3 rats on both the 1.0 and 0.5 ml dosage level had atelectasis (collapse) of one or more lobes of the lung. Fibrotic foreign body nodules were present in all cases.

After 90 days there was chronic foreign body pneumonia in 2 of 3 rats at both dosage levels, fibrotic foreign nodules in 3 of 3 and emphysema in 2 of 3. Chronic inflammatory cell foci and atelectasis were present. Bronchioles were dilated in 2 of 3 rats on both dosage levels and all but one on both dosage levels had some lung hemorrhage.

The final 180 day sacrifice revealed interstitial pneumonia in 4 rats on the 0.5 ml dose. This is a chronic form of pneumonia of interstitial tissue with decrease of the normal lung tissue. Atelectasis was present in the 4 rats on 1 ml and on 1 rat on the 0.5 ml dose of asbestos with the 5 controls normal. Fibrotic foreign body tissue was present in all dosed lungs with none in the controls. Pink hyalin material was found in 2 of 4 lungs from rats on the 1 ml dose while in 3 of 4 there was a bluish homogeneous material evident.

In essence, a total of 10 of 13 rats on the 1 ml dose and 12 of 15 on the 0.5 ml dose had fibrotic foreign body nodules or tissue. There were 3 cases of emphysema on the high dose and 4 on the low dose and 1 in the control. Atelectasis (essentially collapse of lung alveoli) was present in 9 rats on 1 ml and 6 on 0.5 ml of asbestos with none reported in the controls. In general, because of the overwhelming preponderance of effect in the asbestos dosed lungs versus the controls, we have sufficient evidence of damage to warn us to do our best to prevent inhalation of concentrations of asbestos in excess of the Threshold Limit Value proposed for 1970, (Threshold Limit Values of Airborne Contaminants and Intended Changes. Adopted by ACGIH for 1970. American Conference of Governmental Hygienists, 1014 Broadway, Cincinnati, Ohio 45202).

Summary Tables for each of the four sacrifices are included. Detailed pathology reports on each animal are available and copies can be furnished if the need for them arises. A literature review prepared in connection with another request for information is attached although not requested.

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Typed: September 7, 1971 - md

Table 34-19

Tracheal Insufflation to Rats Sacrificed 30 Days After Dosing

		Ml of 1% Solution		
		1	0.5	0.0
TOTAL NUMBER EXAMINED GROSSLY:		3	3	2
LUNG: Number Examined	(M)	3	3	2
Pneumonia	(G)	3	3	0
Hemorrhage	(G)	1	0	0
Pleural adhesions	(G)	1	2	0
Stromal thickening	(M)	3	2	0
Foam cell accumulations	(M)	3	2	0
Granulation tissue foci	(M)	3	3	0
Multinucleated giant cells	(M)	3	2	0
Abscess bronchopneumonia	(M)	0	1	0
Round cell accumulations	(M)	0	0	1
TRACHEA: Number Examined	(M)	3	3	2
Chronic tracheitis	(M)	0	0	1

The following tissues were examined microscopically on all animals: Lung, Liver, Kidney, Heart, Spleen, Adrenal, Thyroid, Parathyroid, Trachea and Esophagus. G = Gross M = Microscopic

Table 34-20

Tracheal Insufflation to Rats Sacrificed 60 Days After Dosing

		Ml of 1% Solution		
		1	0.5	0.0
TOTAL NUMBER EXAMINED GROSSLY:		3	3	2
LUNG: Number Examined	(M)	3	3	2
Hemorrhage	(G)	0	0	1
Pneumonia	(G)	3	3	0
Atelectasis	(G)	0	2	0
Edema	(G)	1	2	0
Hemorrhage	(M)	1	0	0
Atelectasis	(M)	2	2	0
Fibrotic foreign body nodules	(M)	3	3	0
Bronchiolitis fibrosa obliterans	(M)	0	1	0
KIDNEY: Number Examined	(M)	3	3	2
Round cell accumulations	(M)	1	0	0
HEART: Number Examined	(M)	3	3	2
Focal myocarditis	(M)	0	1	0
MUSCLE: Number Examined	(M)	1	0	0
Purulent mass	(G)	1	0	0
Large suppurative process, striated muscle	(M)	1	-	-

The following tissues were examined microscopically on all animals: Lung, Liver, Kidney, Heart, Spleen, Adrenal, Thyroid, Parathyroid, Trachea and Esophagus. G = Gross M = Microscopic

Table 34-21

Tracheal Insufflation to Rats Sacrificed 90 Days After Dosing

		Ml of 1% Solution		
		1	0.5	0.0
TOTAL NUMBER EXAMINED GROSSLY:		3	3	2
LUNG: Number Examined	(M)	3	3	2
Pleural adhesions	(G)	0	2	0
Hemorrhage	(G)	0	2	0
Edema	(G)	3	3	0
Pneumonia	(G)	3	3	0
Chronic foreign body pneumonia	(M)	2	2	0
Fibrotic foreign body nodules	(M)	3	3	0
Emphysema	(M)	2	2	0
Chronic inflammatory cell foci	(M)	3	3	0
Round cell foci	(M)	0	1	1
Atelectasis	(M)	3	3	0
Bronchiectasis	(M)	2	2	0
Stromal thickening	(M)	1	0	0
Hemorrhage	(M)	3	2	0
Inhaled blood	(M)	0	1	0
KIDNEY: Number Examined	(M)	3	3	3
Hydronephrosis	(G)	0	0	1
Hydronephrosis	(M)	0	0	1
Round cell focus	(M)	0	1	0
TRACHEA: Number Examined	(M)	3	3	3
Chronic tracheitis	(M)	2	1	2
HEART: Number Examined	(M)	3	3	3
Myxoid change, interstitium	(M)	0	1	0

The following tissues were examined microscopically on all animals: Lung, Liver, Kidney, Heart, Spleen, Adrenal, Thyroid, Parathyroid, Trachea and Esophagus. G = Gross M = Microscopic

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Table 34-22

Tracheal Insufflation to Rats Sacrificed 180 Days After Dosing

		Ml of 1% Solution		
		1	0.5	0.0
TOTAL NUMBER EXAMINED GROSSLY:		4	6	5
LUNG: Number Examined	(M)	4	6	5
Edema	(G)	2	0	1
Pneumonia	(G)	4	5	1
Atelectasis	(G)	2	0	1
Emphysema	(G)	0	0	1
Emphysema	(M)	1	2	1
Atelectasis	(M)	4	1	0
Inhalation pneumonia	(M)	1	0	0
Interstitial pneumonia	(M)	0	4	0
Abscess bronchopneumonia	(M)	0	0	1
Suppurative bronchiectasis	(M)	1	0	0
Acute bronchitis	(M)	0	0	1
Lymphoid cell accumulations	(M)	3	0	0
Foam cell accumulations	(M)	2	1	3
Fibrotic foreign body tissue	(M)	4	6	0
Granulation tissue foci	(M)	0	1	0
Pink hyalin material	(M)	2	0	0
Bluish homogeneous material	(M)	3	1	0
Numerous mononuclear cells	(M)	1	0	0
Mucoid infiltration	(M)	2	0	0
Proliferation bronchiole epithelium	(M)	0	0	1
LIVER: Number Examined	(M)	4	6	5
Bile duct proliferation	(M)	0	2	1
Round cell foci	(M)	1	0	0
KIDNEY: Number Examined	(M)	4	6	5
Hydronephrosis	(G)	0	1	0
Sand calculi	(G)	0	1	0
Hydronephrosis	(M)	0	1	0
Sand calculi	(M)	0	1	0
Dilated tubules	(M)	0	2	2
Pink casts	(M)	0	2	1
Interstitial nephritis	(M)	0	1	0
Cellular infiltration	(M)	0	1	0
Slight tubular regeneration	(M)	0	1	3
Moderate tubular regeneration	(M)	0	0	1
TRACHEA: Number Examined	(M)	4	6	5
Acute tracheitis	(M)	1	0	0
Chronic tracheitis	(M)	0	4	0

The following tissues were examined microscopically on all animals: Lung, Liver, Kidneys, Heart, Spleen, Adrenal, Thyroid, Parathyroid, Trachea and Esophagus. G = Gross M = Microscopic

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CURRICULUM VITAE

DR E. B. ILGREN
MA (Oxon.), MD, D.Phil. (Oxon.), MACPath., MRCPath (Associate / Neuropathology)

Date of Birth: 13 viii 50

Place of Birth: Philadelphia, Pennsylvania, USA

Nationality: American

Member of Faculty of Biological and Agricultural Sciences and Subfaculty of Biochemistry, University of Oxford

Education

Institute and Location	Degree Field of Study	Year Conferred
Hahnemann Medical University	MD Medicine	1974
American College of Pathology	MACPath Anatomic Pathology (Boards)	1976
University of Oxford	D.Phil (Oxon) Zoology / Botany	1980
Royal College of Pathology UK (Primary)	MRCPath Neuropathology	1982
University of Oxford	MA (Oxon) Biology / Medicine	1983

Research and / or Professional Experience - Chronology

1973 Student Worker, Dept. of Comparative Neuroanatomy, Bryn Mawr College, Bryn Mawr, Penna., USA.

1971 Doctorate of Medicine, The Hahnemann Medical College, Philadelphia, Pennsylvania, USA.

1973 Visiting Resident, The Memorial Hospital for Cancer & Allied Diseases, attached to Dr W.B. Jones, Consultant Gynaecological Surgeon, Trophoblastic Disease Centre.

1974 Assistant Pathologist, The New York Hospital - Cornell University Medical Centre, New York City.

1974 Assistant Forensic Pathologist, Office of the Medical Examiner, The New York University Medical Centre. Dr. M.C. Baden (Sponsor) - Chief Medical Examiner.

1975 Diplomate of the National Board of Medical Examiners (USA).

1975/ 76 Visiting Scientist, Rockefeller Institute of Medical Research. Professor A.C. Braun (Head of Department), Dr E.G. Diacumakos (Laboratory of Cytobiology, Head of Department).

1975 Licensed in Medicine and Surgery, State of New York, No. 124849.

1976 Board Certified, American Board of Pathology. Anatomic Pathology.

Research and / or Professional Experience - Chronology

1976 Visiting Worker. The Imperial Cancer Research Fund. London Prof. R.A. Willis /Dr L.S.C. Pang

1977 Visiting Scientist (Univ. South Calif.. San Diego Zoo. Soc. Hospital. Dept. Path. Professor K. Benirschke

1977 Travelling Research Fellow (International Union Against Cancer) - Geneva, Switzerland.

1976/77 Postdoctoral Research Fellow (International Agency for Cancer Research/World Health Organisation). Dept. of Zoology. University of Oxford. Professor R.L. Gardner. FRS - Sponsor.

1976/79 Special Postdoctoral Research Fellow (American Cancer Society - spf-14). Lab. Experimental Mammalian Embryology, Univ. of Oxford. Prof. R.L. Gardner, FRS.

1978/79 Research Fellow (NIH). Dept. of Zoology. Lab. Experimental Mammalian Embryology, Univ. of Oxford. Prof. R.L. Gardner, FRS

1979/80 Visiting Scientist (NIH), Sir William Dunn School of Pathology, Univ. of Oxford

1979/80 Research Fellow (NIH), Botany School. Lab.of Cytology, Univ. of Oxford. Prof. F. Whatley, FRS / Dr C. Vosa

1980 D.Phil. (Oxon), Dept. of Zoology & Botany, Univ. of Oxford. Member. St Edmund Hall.

1980/81 Honorary Senior Registrar, Neuropathology, The Radcliffe Infirmary, Oxford.

1980/81 Research Fellow (NIH), Dept. of Neuropathology, The Radcliffe Infirmary, Oxford.

1982 Senior Registrar, Neuropathology, The Radcliffe Infirmary, Oxford.

1982 Pathologist to H.M. Coroner, Mr N.G. Gardiner (Oxfordshire).

1982 General Medical Council, Full Registration.

1983 (Locum) Consultant Neuropathologist, The Radcliffe Infirmary, Oxford, June 3-17, 1983.

1983 Subfaculty Member, Department of Biochemistry, University of Oxford.

1984 Faculty Member, Biological Sciences, University of Oxford.

1984 Co-recipient, Health & Safety Executive project grant award: co-recipient with Prof P. Shubik, "Iron Chelation: An attempt to develop a possible therapy for those at risk from exposure to asbestos by inhibiting formation, persistence, and effects of asbestos bodies *in vivo*".

1985 Recipient, Oxford Area Health Authority / District Research Grant Award "Embryonic regulation of neural neoplasia".

PUBLICATIONS

33. Ilgren, E.B. and Stiller, C. (1987) Review article: Cerebellar astrocytomas. Part II. Pathologic features indicative of malignancy. Clin. Neuropath., 6: 201-214

34. Ilgren, E.B. (1988). Multi System Atrophy: Possible central aetiology of the laryngeal dysfunction and the role of lymphocytic inflammation. Arch. Suisses Neuro. Psych. 139: 75.

35. Ilgren, E.B. (1989). Mesothelioma Threshold. In: Effects of Mineral Dusts on Cells. (Mossman, B. and Begin, R.). Springer-Verlag, Heidelberg, 1989, vol. H.30, 455-464.

36. Ilgren, E. and Wagner, J. (1989). The value of experimental animals in analyzing the hazards of exposure to mineral fibres. II. Consideration of natural background incidence of mesotheliomas and nonphysiological intraperitoneal technique. Submission to the German MAK Commission on Mineral Fibre Safety. ISBN [0-9516359-0-5].

37. Ilgren, E.B. and Wagner, J.C. (1991). Background incidence of mesothelioma: Animal and human evidence. Reg. Tox. Pharm., 13: 133-149

38. Ilgren, E.B. and Browne, K. (1991). Asbestos-related mesothelioma: Evidence for a threshold in humans and animals. Reg. Tox. Pharm., 13: 116-132

39. Brown, R., Davis, J., Douglas, D., Gruber, U., Hoskins, J., Ilgren, E., Johnson, N., Rossiter, C. and Wagner, J. (1991). Carcinogenicity of the insulation wools: Reassessment of the IARC Evaluation. Reg. Tox. Pharm., 14: 12-23

40. Ilgren, E.B. (1991) The potential health effects from low dose exposure to tremolite in vermiculite: An overview of the Libby, Enoree, and the Palaora experience. [Abstract]. Fourth International Conference, Environmental Lung Disease, 25-28. Sept 1991 Montreal.

41. Ilgren, E. (1992) Potential hypersusceptibility to the induction of mesotheliomas in hamsters exposed to ceramic fibres: Theoretical biological mechanisms. Reg. Tox Pharm [submitted]

Books:

Ilgren, E [1991] Initiation and Promotion in Skin or Liver Neoplasia: A 65 year annotated bibliography of international literature. CRC press. 928 pp. ISBN - 0-8493-4407-7

Ilgren, E [1993] Mesotheliomas of Animals: A comprehensive, tabular compendium of the world's literature. CRC press. 356 pp. ISBN - 0-8493-4308-9

Employment

Dates (month & year) From	To	Name and address of sponsor/employer	Position
April 1987	July 1989	Imperial Cancer Research Fund	Research Officer (Hon.) Clinical Trials Study Unit. Univ. Oxford.
June 1989	Present	University of Oxford, St Edmund Hall	Member, Faculty (Biological Sciences). Subfaculty (Biochemistry)

Research Experience

The basic and clinical aspects of the tumour problem and neurodegenerative disease have been persistent themes of the research conducted to date.

Rockefeller University, 1975 - 1976:

"Control of Trophoblastic HCG Secretion: Gene Mapping": EBI, in collaboration with Dr E.G. Diacumakos (Rockefeller University) and Dr W.B. Jones (Memorial Hospital for Cancer), was the first to grow choriocarcinomas *in vitro* without recourse to animal hosts, work conducted in a combined clinical and experimental study of the relationship between HCG secretion and malignant trophoblastic growth. The results of this study suggested that the secretory activity and cellular proliferation of malignant trophoblast may, at times, be dissociated phenomena *in vitro*, a conclusion confirmed by observations made, on rare occasions, *in vivo*. Since these experiments were originally aimed at mapping the HCG gene via cell hybridisation, EBI also mastered techniques, developed by Dr Diacumakos, for microsurgical cell fusion, chromosome extraction, nuclear and cytoplasmic cell injection, and single cell cloning as well as methods needed for the cytogenetic analysis of such cloned cell lines. Whilst at Rockefeller, EBI also had numerous discussions with Prof. Armin Braun on Epigenetic theory, tumour suppression, and recovery. These were based on Prof. Braun's Crown Gall Teratoma Model System and laid the foundation for some of EBI's future experiments.

Imperial Cancer Research Fund, Tumour Reference Collection (TRC) and the San Diego Zoological Research Hospital (SDZ), 1976:

"Comparative Pulmonary Neoplasia": EBI made an extensive histopathological review of a large series of pulmonary tumours found in captive wild animals (SDZ) and various domestic species (TRC), the results confirming previous reports that most animal lung tumours are of the alveolar type, i.e. the one not generally seen in association with smoking.

Oxford University, Dept. Zoology, 1976 - 1981:

"Control of Trophoblastic Cellular Division": The perplexities of working with a malignant tissue that, even in their normal state, behave in a manner similar to tumour tissues, led EBI to pursue studies of normal trophoblastic growth and developmental mechanisms generally thought to be related to neoplasia. Such studies were pursued in Oxford in the microsurgery laboratory of Prof. R.L. Gardner, a leading experimental mammalian embryologist. These studies employed microsurgical injection, dissection, enzymatic separation, and / or tissue culture of pre- and post-implantation embryos and their constituent tissues. The latter were analysed biochemically, histochemically, cytologically, cytogenetically, histologically, and cytophotometrically using, in some cases, methods developed by the EBI. Embryo transfer and tumour inoculation methods were also employed in these studies.

Oxford University, Dept. of Botany, 1980 - 1981:

"Cytological and Cytogenetic Analysis of Polyploid Cells": Preparations of trophoblastic giant cells were analysed cytologically, cytogenetically, and cytophotometrically in the Botany School under the supervision of two eminent cytologists, Dr C.G. Vosa and Dr F.A.L. Clowes. These analyses were primarily directed at assessing DNA synthesis and chromosome structure.

Research Experience

Oxford University, Sir William Dunn School of Pathology, 1980-1981:

"Regulation and Suppression of the Tumorous State: Analysis of Heterotopic Embryonal Carcinoma (EC) cell-derived Chimeric tissues *In Vitro* and *In Vivo*": Embryonal carcinoma (EC) cell-derived mouse chimaeras were produced by EBI using Prof. Gardner's blastocyst injection methods and the mature chimaeric tissues were subsequently dissected into their constituent parts, ectopically transferred to syngeneic hosts, and observed in host sites for tumour growth of donor cell origin. Since ectopically transferred, chimaeric, placental tissues were the only ones to form tumours, it was concluded that placental, in contrast to somatic tissues, lacked the ability to suppress the tumorous phenotype.

Oxford University, Dept. of Neuropathology, The Radcliffe Infirmary, 1981-1985.

"Embryonic Regulation of Neural Neoplasia": Discussions with Prof. Armin Braun subsequently led EBI to ask if tumour cell types other than those of EC cell origin, could colonise the embryo and, in turn, be suppressed or regulated by the embryonic environment. Since it was likely that this would occur with 'developmentally synchronised', donor-host cell combinations, e.g. when neuroblastomas were injected into the normal neuroblastic tissues of the embryo, a microsurgical method was developed that could inject marker substances and cells into the brain of the developing mouse embryo *in utero*.

"Unusual associations between different tumour types and hamartomatous dysgenetic disorders of neural crest (e.g. intra-cranial melanoma and Naevus of Ota) or non-neural crest (e.g. Tuberous Sclerosis with Sarcomas and Adenocarcinomas) origin": EBI was to first to provide evidence to suggest that the NF gene in Neurofibromatosis may not only predispose neural crest-derived Schwann cells to undergo malignant change but also may affect, in a similar way, non-neural crest, glial derivatives of the cerebellum.

"Clinical Pathological Correlation Studies": The largest series of ependymomas, cerebellar astrocytomas, medulloblastomas, and craniopharyngiomas were analyzed to determine clinical and pathological prognostic indices.

"Involvement of Nucleus Ambiguus and Chronic Inflammation in MultiSystemic Atrophy (MSA)": EBI was the first to demonstrate chronic inflammation in the Shy-Drager Syndrome (Multi System Atrophy with Autonomic Involvement) and to confirm upper motor neuron loss within the nucleus ambiguus by histologically reviewing serial sections of the brainstems of more than 20 MSA patients, perhaps the largest collection in the world.

"Neuroepidemiological Studies: Mercury containing pesticides - An assessment of the exposure, neuromuscular function, and neuropathological status of seed dressing merchants and agricultural workers": A multidisciplinary investigation to assess the exposure, neuromuscular function, and possible neuropathological changes in seed dressing merchants and workers exposed to mercury-containing pesticides in Oxfordshire was outlined though not conducted. The investigation had received the approval of the University Dept. of Neurology, The Radcliffe Infirmary, Oxford (Prof. B. Matthews); the National Hospital for Nervous Disease, Dept. of Neurophysiology, Queen's Square, London; DHSS St Bartholomew's Medical College, London (Prof. A.D. Dayan (Toxicology) and Prof. N.J. Wald (Epidemiology)); the Oxford Scanning Proton Microprobe Group, Dept. of Nuclear Physics, University of Oxford (Dr F. Watt); and the Dept. of Neuropathology, The Radcliffe Infirmary, Oxford Dr E.B. Ilgren). Extensive discussions were conducted with the Health and Safety Executive (UK) (Dr D. Gompertz); the Pesticide Regulation Dept., Ministry of Agriculture, Food & Fisheries (MAFF) (Dr J.M.

Sly); Advisory Committee on Pesticide Use (UK) (Prof R. Kilpatrick); the Myaesthenia Gravis Foundation (USA) (Dr E. Lambert); the Executive Director, British Agrochemical Association (Mr I. Abbott); the Product Safety and Plant Protection Division, Imperial Chemical Industries (UK); Dr G. Worthing, Advisor, Pesticide Safety, British Crop Protection Council; Dr S. Logan, Medical Advisor, Shell International (The Hague); Dr E. Burgess, Medical Advisor, Dow Chemical (UK); Dr G. Bonsall, Plant Protection Advisor, Fisons Boots Co. (UK); Mr B. Syde, Dalgety (UK); and Weed Research Organization (Oxford).

Oxford University, Dept. of Nuclear Physics, 1984:

"Nuclear Proton Microprobe Study of CNS Tissues": In collaboration with the Oxford Proton Microprobe Group, EBI was the first to demonstrate the endogenous elements of the human brain with the proton microprobe thereby generating an 'elemental map' which closely corresponded to the normal architecture of that tissue.

Oxford University, 1985 - 1987:

"Asbestos Body / Chelator Treatment Project". With the assistance of Prof. P. Shubik, EBI investigated methods of modifying the inflammation secondary to crocidolite and the zeolite, erionite to see if the inhibition of ferruginous body formation could suppress neoplasia.

Oxford University, Imperial Cancer Research Fund, Clinical Trials Studies Unit, 1985 - 1989:

"Initiation and Promotion in Skin or Liver Neoplasia: A 65 year annotated Bibliography of International Literature": The terms tumor "initiator" and "promoter" have engendered much confusion particularly within the regulatory agencies responsible for their control. Because of this, Prof. P Shubik was asked by the American Industrial Health Council (AIHC), the National Cancer Institute (NCI), and the Health and Welfare Department of Canada to attempt to clarify such confusion and reach a greater consensus on the present day meaning and definition of such terms. Prof Shubik then asked EBI to comprehensively compile and critically review every original whole animal study ever performed that was claimed to be related to initiation and promotion in the skin or liver of any species as a first step towards achieving this goal. The first 13 months of the project was supported by the AIHC (ca. $50, 000) but was completed with the support of the Imperial Cancer Research Fund (ca $200, 000) under the supervision of Dr Richard Peto, FRS over the ensuing 32 months. The vastness of the literature reviewed for the project (over 2000 original studies from 200 different journals in 9 languages) is reflected in the fact that the articles upon which the study was primarily based occupied more than 25 feet of shelf space. The phenomenon of initiation and promotion has substantially influenced past and current theories of human cancer and continues to be the focus of much research. The project, described in a 928 page (microtext) compendium published by CRC press in 1991, attempts to make the relevant experimental evidence readily accessible. A greater aim was to bring together studies that relate importantly to phenomena that have been of increasing concern to those involved with environmental protection legislation namely events pertaining to possible human exposures to agents with delayed cancer causing effects and to the potential synergism between different agents. Overall, however, one must be cautious about overinterpreting the animal data found in many studies that claim to investigate initiation and promotion since the majority have utilized nonphysiological models, unduly sensitive strains, and excessive exposures often in excess of the maximum tolerated dose.

Research Experience

"Irreversibility of the Initiated State": Early initiator - promoter studies claimed that initiation was irreversible ie that a dose of carcinogen insufficient to induce tumors was still sufficent to produce a cancer risk that would remain unabated for life. However, this concept (which forms the basis of the no - threshold concept) defies biological sense in the light of our present day knowledge especially on repair mechanisms. Thus, in order to demonstrate that the studies subsequent to the earlier ones fail to support the concept of irreversibility, all of the studies which claimed to test the reversibility of initiation were therefore assembled and critiqued. The results were then presented as invited lectures, to universities (Bryn Mawr College), industrial centres (ICI, Eli Lilly), and to government (FDA). The work was supported (ca $10,000) by the DHSS, Dept. of Toxicology, London, Dr Barbara MacGibbon and Dr John Steadman.

Oxford University, Faculty, Biological Sciences, St. Edmund Hall, 1989 to present.

"Mesotheliomas of Animals: A Comprehensive, Tabular Compendium of the World's Literature":EBI produced the first book to comprehensively compile the diverse, disparate literature on animal mesotheliomas. The compendium presented evidence in support of the existence of so - called "background" or nonasbestos related mesotheliomas as well as the threshold concept.

"Mesothelioma Background: Nonasbestos related tumors":In collaboration with Dr J C Wagner, EBI assembled, analyzed, and published the animal and human evidence which supports the view that mesotheliomas may be caused by agents other than asbestos. EBI is presently extending this work by assessing the possible presence of fibrous zeolite - related disease in the United States.

"Mesothelioma Threshold": In collaboration with Dr K Browne, EBI assembled, analyzed, and published the animal and human evidence that supports the view that the doses of agents required to induce mesothelioma display no observable effect levels or threshold. This was done primarily for the two major commercial amphiboles, amosite and crocidolite. EBI is presently extending these studies to tremolite, the common contaminant of chrysotile, vermiculite, and talc. In particular, the pathology Libby vermiculite / tremolite miners and millers with lung cancer and mesothelioma being analyzed for publications in conjunction with lung burden, hygiene, and other occupational exposure data.

"Chrysotile: Biological Effects of Naturally Occuring Short Fibre Preparation from Calidria":In collaboration with Dr Kent Pinkerton and Dr E C Mc Connell, EBI is finalizing the results obtained from a long term bioassay using two standard long fibre chrysotile preparations, UICC / B and Jeffrey, and the short fibre preparation from Calidria, California.

"Amosite Related Mesothelioma": EBI is producing a critical overview of the mesotheliomas noted in association with amosite exposure. He is also attempting to set up a UK based, multidisciplinary study to update and finalize both the Germiston and the Uxbridge amosite factory cohorts with a particular view towards "profiling", in detail, the mesothelioma cases.

Memberships

St Edmund Hall (Middle Common Room). Univ. Oxford	1976
British Society Development Biologists (BSDB)	1979
Tuberous Sclerosis Association (UK)	1981
Neurofibromatosis Foundation (USA and UK)	1981
Royal College of Pathology (Asso.)	1981
British Society of Neuropathologists (BSN)	1981
Neurosciences Specialty Group, University of Oxford	1981
Oxford Proton Microprobe Group, Nuclear Physics, Univ. Oxford	1983
British Medical Association (BMA)	1984
Subfaculty of Biochemistry, Univ. of Oxford	1984
Faculty of Biological Sciences, Univ. of Oxford	1984
Oxford University Computer Centre Users Group	1985
International Fibre Safety Group (IFSG)	1990
Technical Services Group (TSG), Wash., DC	1991
American Medical Association (AMA)	1992
Pennsylvania Medical Society	1992
Montgomery County Medical Society	1992

Awards

American Cancer Society Special Postdoctoral Award (Control of Trophoblastic Growth)	June 1976 - June 1978
Intl. Agency for Cancer Research Postdoctoral Award (Control of Trophoblastic Growth)	Dec. 1976 - June 1977
Intl. Union Against Cancer (ICRETT) Award (Comparative Pulmonary Oncology)	June 1977 - July 1978
National Inst. Health Postdoctoral Training Award (Control of Trophoblastic Growth)	June 1978 - Sept. 1980
Medical Research Council Project Growth Award (Embryonic Regulation of Neural Neoplasia)	April 1982 - Sept. 1984
Health & Safety Executive Project Grant Award (Potential Therapy ... Asbestosis)	Sept. 1984 - July 1985
Oxford District Research Grant Award (Embryonic Regulation of Neural Neoplasia)	Nov. 1984 - July 1985
American Industrial Health Council Award (Tumor Promotion Project)	Aug. 1985 - Feb. 1986
Imperial Cancer Research Fund Award (Tumor Promotion Project)	Mar. 1987 - Aug. 1989
W R Grace & Co. Research Award (Health Effects of Tremolite Project)	Aug. 1990 - Sept. 199[illegible]

Doctoral Dissertation - Summary

1. Control of trophoblastic proliferation in early postimplantation mouse development: Several conditions are needed to promote the growth of postimplantation mouse trophoblast. Firstly, an appropriate tissue shape and close cell contacts are needed to suppress the trophoblastic giant cell transformation and maintain these tissues in a largely diploid, non-giant state. Moreover, since an appropriate tissue shape cannot, on its own, promote trophoblastic cell division, ICM-derivatives, e.g. extraembryonic endoderm and / or mesoderm, are probably also needed to stimulate normal trophoblastic growth.

2. Control of trophoblastic proliferation in the guinea pig: The growth of guinea pig trophectoderm and its early postimplantation derivatives appear to be under ICM control. Thus, the multilayered attachment cone of the guinea pig blastocyst is probably not due to an ICM-independent proliferation of trophectoderm but is most likely derived from a transient accumulation of abembryonic trophectodermal cells.

3. Origin of the trophoblastic giant cells: binucleation: In the mouse, postimplantation trophoblast appears to become giant through a binucleate phase. Thus, the growth of mouse trophectoderm occurs, to a certain extent, in a manner similar to the polyploidisation of mouse liver.

4. Origin of the trophoblastic giant cell: multinucleation: The finding of multinucleates in pure, trophectoderm-derived tissues suggests that trophoblastic multinucleation, at least in the mouse, does not depend upon prior contact with mesenchyme or mesenchymal tissues. Multipolar, polyploid mitotic figures were found in mouse decidua, tissues known to contain multinucleates similar to the ones seen in the trophoblastic derivatives of ruminants. This suggests that the multinucleate cells seen in some forms of trophoblast may arise in a manner similar to those found in rodent decidual tissues, i.e. via a multipolar mitosis without concomitant cytokinesis.

5. Mechanism by which DNA accumulates during the trophoblastic giant cell transformation: Polyploid metaphases were found within postimplantation mouse trophoblast that had chromosomes arranged either randomly or in pairs. This suggests that at least some mouse trophoblastic cells increase their DNA via polyploid mitoic and/or endomitotic cycles.

6. Control of the trophoblastic giant cell transformation: The degree to which trophectoderm-derived cells endoreduplicate their nuclear genome and thus become giant can be influenced by changes in tissue shape and the extent of heterocellular contact. Thus, cell separation, spreading, and stretching appear to promote the giant cell change not only within trophectodermal derivatives but also within tissues known to behave in a manner similar to trophectoderm, namely extraembryonic endoderm. Moreover, the giant cells found in normal and neoplastic extraembryonic tissues are not necessarily terminal and incapable of cell division since they may be found in mitosis either before and / or following mitogenic stimulation. Comparative studies also suggest that the onset of the giant cell change in one extraembryonic membrane may be temporally correlated with the development and / or degeneration of a nearby placental tissue layer.

Invited Lectures

1982 British Neuropathology Society (London) ("Prognostic Indices for Human Ependymomas")

1983 Ludwig Institute for Cancer Research. University of Bern ("Regulation of Neural Neoplasia")

1984 Faculty of Medicine. University of Lausanne ("Regulation of Neural Neoplasia")

1984 Institute for Brain Research. Zurich ("Elemental Mapping of the Human Brain")

1988 Swiss Neuropathology Society (St Moritz) ("Chronic Inflammation and Involvement of the Nucleus Ambiguus in MultiSystem Atrophy")

1989 Alfred P. Sloan Lecture. Bryn Mawr College ("Transplacental Initiation / Postnatal Promotion: Fetal Risk from Maternal Exposure")

1989 ICI (UK) - Central Toxicology Laboratory ("Complete Carcinogenesis: Ultraviolet Light and Chemical Carcinogens")

1989 ICI (Americas) - Wilmington, Delaware ("Initiation Irreversibility")

1989 Food and Drug Administration - Toxicology (Wash. D.C.) ("Initiation Irreversibility: Epidermal Studies")

1990 Food and Drug Administration - Toxicology (Wash. D.C.) ("Initiation Irreversibility: Non-Epidermal Studies")

1990 Eli Lilly - Central HQ, Ind., Ind. ("Carcinogenesis / Promotion Project")

1991 American College of Chest Physicians. Environmental Lung Disease. Fourth International conference. 25 - 28 Sept 1991 ("The potential health effects from low dose exposure to tremolite in vermiculite: An overview of the Libby, Enoree, and the Palabora experience".) (Montreal).

1991 International Congress of Occupational Health and Safety. (Montreal) 7 - 9 Sept 1991 ("Background mesotheliomas")

1991 Defense Research Institute. Asbestos Medicine Seminar (Scottsdale, Arizona) 20 - 23 Oct 1991 Faculty lecture ("Mesothelioma threshold")

1992 Dow Chemical Company (Saginaw, Michigan) 12 July 1992 ("Mechanisms of Fibre related disease")

1992 International Agency for Research on Cancer. World Health Organization (Lyon, France). Biopersistence of mineral fibres ("Potential hypersusceptibility to the induction of mesotheliomas in hamsters exposed to ceramic fibres: theoretical biological mechanisms")

1993 Lead Industries Association (LIA) ("Lead and cancer") 11 October 1993, St Louis, Mo.

PUBLICATIONS

1. Ilgren, E.B., Tang, C.K. and Thorbjarson, L. (1976). Cystadenocarcinoma of the pancreas. New York State Journal of Medicine 76: 548-550.

2. Ilgren, E.B., Symchyk, P.S. and Redo, S.F. (1977). Pneumoperitoneum without rupture viscus in the neonate: a case report and review of the literature. J. Ped. Surgery. 12: 537-540.

3. Ilgren, E.B. (1980). Polyploidization of extraembryonic tissues during mouse embryogenesis. J. Emb. exp. Morph. 59: 103 - 111.

4. Ilgren, E.B. (1980). The control of trophoblastic growth in the guinea pig. J. Emb. exp. Morph., 60: 405-418.

5. Ilgren, E.B. (1981). The control of trophoblastic giant-cell transformation in the mouse: homotypic cellular interactions and polyploidy. J. Emb. exp. Morph. 62: 183-202.

6. Ilgren, E.B. and Little field, J.W. (1981). The extra embryonic endodermal differentiation and polyploidization of embryonal carcinoma cells in vitro. Differentiation 19: 115-20.

7. Ilgren, E.B. (1981). The in vitro morphogenesis of the guinea-pig egg cylinder. Anat. Emb., 163: 351-365.

8. Ilgren, E.B. (1981). Placental polyploidy: a comparative study in the mouse and the guinea-pig. Placenta, 2: 333-342.

9. Ilgren, E.B. (1981). The initiation and control of trophoblastic growth in the mouse: binucleation and polyploidy. Placenta, 2: 317-332.

10. Ilgren, E.B., Griner, L., Benirschke, K. and Pang, L.S.C. (1982). A comparative study of pulmonary tumors from the San Diego Zoological Gardens and the Tumour Reference Collection, Imperial Cancer Research Fund, London. Pathology Annual, 17: 331-351.

11. Pearson, J., Ilgren, E.B. and Spriggs, A.J. (1982). Lymphoma cells in cerebrospinal fluid confirmed by chromosome analysis. J. Clin. Path., 35: 1307 - 1311.

12. Ilgren, E.B., Brings, M. and Aynesley-Green (1983). Precocious puberty in a 3-year-old girl associated with a parasellar ganglionic hamartomas. Clin. Neuropath., 2: 95-98.

13. Ilgren, E.B. and Vaux, D. (1983). The comparative pathology of teratomas. Cancer Surveys, 2(1):209 - 215.

14. Ilgren, E.B. (1983). Review article: Control of trophoblastic growth. Placenta, 4: 307-328.

15. Ilgren, E.B., Evans, E.P. and Burtenshaw, M.D. (1983). Origin of the multinucleate decidual cell of the mouse. Cytologia, 48: 313-322.

16. Ilgren, E.B. (1983). Cardiomyogenic differentiation and teratocarcinoma formation in EC-derived chimaeric placentae. Placenta, 4: 415-422.

17. Sagar, H., Ilgren, E.B. and Adams, C.B.T. (1983). Nevus of Ota associated with meningeal melanosis and intracranial melanoma. J. Neurosurg., 58: 280-283.

PUBLICATIONS

18. Ilgren, E.B. and Hemachudha, S. (1983). Dissecting aneurysm of the pulmonary artery associated with brain abscess: a case report. Clint. Neuropath., 2:179-181.

19. Ilgren, E.B., Stiller, C., Steckel, M., Silverman, P. and Hughes, J.T. (1984). Ependymomas:a clinical and pathological study. Part I. Biological Features. Clin. Neuropath., 3: 113-121.

20. Ilgren, E.B., Stiller, C., Steckel, M., Silberman, P., Hughes, J.T. and Kaye, A. (1984). Ependymomas: a clinical and pathological study. Part II. Survival Features. Clin. Neuropath., 3:122-127.

21. Ilgren, E.B., West Moreland, D. and Adams, C.B.T. (1984). Cerebellar mass caused by *Candida* species. J. Neurosurg., 60: 428-430.

22. Ilgren, E.B. and West Moreland, D. (1984). Tuberous sclerosis: Unusual associations in four cases. J. Clin. Path., 37: 272-278.

23. Ilgren, E.B. and Teddy, P.J. (1984). Chemodectoma of the Cauda Equine: a case report. Clin. Neuropath., 3:148-152.

24. Ilgren, E.B., Teddy, P.J., Vafidis, J., Briggs, M. and Gardiner, N.G. (1984). Clinical and pathological studies of brain injuries in horse riding accidents: a description of cases with a warning to the unhelmeted. Clin. Neuropath., 3: 253-259.

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28. Ilgren, E.B., Saxton, D.G., Jones, A.E. and Duckworth, S. (1985). A potential microneurosurgical method for the manipulation of the developing nervous system of the postimplantation mouse embryo *in utero*. I. Injection methods. J. Neurosci. Methods, 13:191-197.

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PUBLICATIONS

33. Ilgren, E.B. and Stiller, C. (1987) Review article: Cerebellar astrocytomas. Part II. Pathologic features indicative of malignancy. Clin. Neuropath., 6: 201-214

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38. Ilgren, E.B. and Browne, K. (1991). Asbestos-related mesothelioma: Evidence for a threshold in humans and animals. Reg. Tox. Pharm., 13: 116-132

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41. Ilgren, E. (1992) Potential hypersusceptibility to the induction of mesotheliomas in hamsters exposed to ceramic fibres: Theoretical biological mechanisms. Reg. Tox Pharm [submitted]

Books:

Ilgren, E [1991] Initiation and Promotion in Skin or Liver Neoplasia: A 65 year annotated bibliography of international literature. CRC press. 928 pp. ISBN - 0-8493-4407-7

Ilgren, E [1993] Mesotheliomas of Animals: A comprehensive, tabular compendium of the world's literature. CRC press. 356 pp. ISBN - 0-8493-4308-9

PLF Ilgren
DMB EX 2
6/21/94 DMB

E. B. ILGREN, MA. MD. DPhil. MACPath. MRCPath (Assoc./Neuropathology)
FACULTY OF BIOLOGICAL SCIENCES, SUBFACULTY OF BIOCHEMISTRY,
UNIVERSITY OF OXFORD, ST EDMUND HALL, OXFORD
AND
RESEARCH OFFICER, IMPERIAL CANCER RESEARCH FUND,
CLINICAL TRIALS STUDY UNIT
NUFFIELD DEPARTMENT OF CLINICAL MEDICINE,
RADCLIFFE INFIRMARY, OXFORD.

Exhibit 1
DMB

CURRICULUM VITAE

Dr E. B. Ilgren, MA (Oxon), MD, D.Phil.(Oxon),
MACPath, MRCPath (Associate/Neuropathology)

Date of Birth: 13.viii.50

Place of Birth: Philadelphia, Pennsylvania, USA

Nationality: American

PRESENT POSITION

Member of Faculty of Biological and Agricultural Sciences and
Subfaculty of Biochemistry, University of Oxford

Research Officer, Imperial Cancer Research Fund,
Cancer Studies Unit
University of Oxford
Nuffield Department of Clinical Medicine
The Radcliffe Infirmary
Oxford OX2 6HE

EDUCATION

INSTITUTION AND LOCATION	DEGREE	YEAR CONFERRED	FIELD OF STUDY
Hahnemann Medical University	MD	1974	Medicine
American College of Pathology	MACPath	1976	Anatomic Pathology
University of Oxford	D.Phil (Oxon)	1980	Zoology/Botany
Royal College of Pathology UK (Primary)	MRCPath	1982	Neuropathology
University of Oxford, Oxford, UK	MA (Oxon)	1983	Biology/Medicine

RESEARCH AND/OR PROFESSIONAL EXPERIENCE - CHRONOLOGY

1973 Student Worker, Dept. of Comparative Neuroanatomy, The Bryn Mawr College, Bryn Mawr, Pennsylvania, USA.

1974 Doctorate of Medicine, The Hahnemann Medical College, Philadelphia, Pennsylvania, USA.

1973/74 Visiting Resident, The Memorial Hospital for Cancer & Allied Diseases, attached to Dr. W.B. Jones, Consultant Gynaecological Surgeon, Trophoblastic Disease Centre.

1974 Assistant Pathologist, The New York Hospital - Cornell University Medical Centre, New York City.

1974 Assistant Forensic Pathologist, Office of the Medical Examiner, The New York University Medical Centre, Dr. M.C. Baden (Sponsor) - Chief Medical Examiner.

1975 Diplomate of the National Board of Medical Examiners (USA)

1975/76 Visiting Scientist, Rockefeller Institute of Medical Research, Professor A.C. Braun (Head of Department), Dr. E.G. Diacumakos (Laboratory of Cytobiology, Head of Department).

1975 Licensed in Medicine and Surgery, State of New York, No. 124849.

1976 Member, American College of Pathologists, Board Certified, American Board of Pathology, Anatomic Pathology.

1976 Visiting Worker, The Imperial Cancer Research Fund, Dept. of Pathology, London - Professor R.A. Willis and Dr. L.S.C. Pang (Head of Department).

1977 Visiting Research Scientist (Univ. of South California at San Diego, San Diego Zoological Society and Dept. of Pathology, Professor K. Benirschke - Sponsor.

1977 Travelling Research Fellow (International Union Against Cancer) - Geneva, Switzerland.

1976/77 Postdoctoral Research Fellow (International Agency for Cancer Research/World Health Organisation), Dept. of Zoology, University of Oxford, Professor R.L. Gardner (FRS) - Sponsor.

1976/79 Special Postdoctoral Research Fellow (American Cancer Society -spf-14), Laboratory of Experimental Mammalian Embryology, Univ. of Oxford, Professor R.L. Gardner, (FRS).

1978/79 Research Fellow (NIH), Dept. of Zoology, Laboratory of Experimental Mammalian Embryology, Univ. of Oxford, Professor R.L. Gardner (FRS) - Sponsor.

1979/80 Visiting Research Worker (NIH), Sir William Dunn School of Pathology, Univ. of Oxford, Oxford.

1980 D.Phil (Oxon), Dept. of Zoology & Botany, Univ. of Oxford, Member of St. Edmund Hall.

1979/80 Research Fellow (NIH), Botany School, Laboratory of Cytology, Univ. of Oxford, Oxford, Professor F. Wheatley, FRS, and Dr. C. Vesa (Sponsors).

1980/81 Honorary Senior Registrar, Neuropathology, The Radcliffe Infirmary, Oxford.

1980/81 Research Fellow (NIH), Dept. of Neuropathology, The Radcliffe Infirmary, Oxford.

1981/82 Senior Registrar, Neuropathology, The Radcliffe Infirmary, Oxford.

1982 Pathologist to H.M. Coroner, Mr. N.C. Gardiner (Oxfordshire).

1982 General Medical Council, Full Registration.

1982 Recipient, MRC Project grant award "Embryonic Regulation of Neural Neoplasia."

1983 MA (Oxon) - Status: Primary, MRCPath (Neuropathology).

1983 (Locum) Consultant Neuropathologist, The Radcliffe Infirmary, Oxford, June 3-17, 1983.
1983 Member, Department of Biochemistry, University of Oxford, Subfaculty.
1983 Member, Oxford Nuclear Physics Proton Microprobe Users Group.
1984 Member, Faculty of Biological Sciences, University of Oxford.
1984 Co-recipient. Health & Safety Executive project grant award: co-recipient with Professor P. Shubik, "Iron Chelation: An attempt to develop a possible therapy for those at risk from exposure to asbestos by inhibiting formation, persistence, and effects of asbestos bodies in vivo."
1985 Recipient. Oxford Area Health Authority/District Research Grant Award "Embryonic regulation of neural neoplasia."

Dates (month & year) From	To	Name and address of employer (block letters)	
June 1974	June 1976	New York Hospital/Cornell University Med. Centre	Assistant Pathologist, Anatomic Path. (Registrar/Sr. Reg. Grade)
June 1976	Sep. 1976	Office of the Medical Examiner (NY) New York Univ.Med.Centre	Assistant Pathologist, Forensic Path. (Sr. Reg./Consultant Grade)
Dec. 1976	June 1977	Intl. Agency for Research on Cancer/World Health Org. (Lyon)	Research Fellow, Embryology, Cell Biology, Oncology (also see 'Research Experience')
June 1977	June 1978	American Cancer Soc. (NY)	Research Fellow, Embryology, Cell Biology, Oncology
June 1978	Sep. 1980	NIH (Bethesda, MD)	Research Fellow, Embryology, Cell Biology, Oncology
Sep. 1982	June 1985	HM Coroner (Oxford)	Forensic Neuropathology. (Senior Registrar/Consultant (Locum) Grade)
Sep. 1980	June 1985	National Health Service (UK)	Neuropathologist (Senior Registrar/ Consultant (Locum) Grade)
Dec. 1984	Dec. 1985	Health & Safety Executive (London)	Research Pathologist (see 'Research Experience')
July 1984	Apr. 1987	Univ. of Oxford, Research Fellow/Faculty Member/ Biological Sciences	Toxicologist
		Sponsors American Industrial Health Council (AIHC) International Life Sciences Institute (ILSI) Certified Color Manufacturers Association (CCMA) Flavor Extract Manufacturers Association (FEMA) Research Institute for Fragrance Materials (RIFM) Chemical Manufacturers Association (CMA) Procter & Gamble Coca-Cola Company Pepsico	
Apr. 1987	Present	Univ. of Oxford/Imperial Cancer Res. Fund, Cancer Studies Unit, Nuffield Dept. of Clinical Medicine Radcliffe Infirmary, Oxford	Research Officer

Details of training, post qualification research periods

1973/74	Visiting Resident, The Memorial Hospital for Cancer & Allied Diseases, attac. to Dr W.E. Jones, Consultant Gynaecological Surgeon, Trophoblastic Disease Centre.
1974	Assistant Pathologist, The New York Hospital - Cornell University Medical Centre, New York City.
1975	Assistant Forensic Pathologist, Office of the Medical Examiner, The New York University Medical Centre, Dr M.C. Baden (Sponsor) - Chief Medical Examiner.
1975	Diplomate, National Board of Medical Examiners (USA).
1975/76	Visiting Scientist, Rockefeller Institute of Medical Research, Professor A.C. Braun (Head of Department), Dr E.C. Diacumakos (Laboratory of Cytobiology, Head of Department).
1975	Licensed in Medicine and Surgery, State of New York, No. 124849.
1976	Visiting Worker, The Imperial Cancer Research Fund, Dept. of Pathology, London - Professor R.A. Willis and Dr L.S.C. Pang (Head of Department).
1977	Visiting Research Scientist (Univ. of South California at San Diego, San Diego Zoological Society and Dept. of Pathology, Professor K. Benirschke - Sponsor.
1977	Travelling Research Fellow (International Union Against Cancer) - Geneva, Switzerland.
1976/77	Postdoctoral Research Fellow (International Agency for Cancer Research/World Health Organisation), Dept. of Zoology, University of Oxford. Professor R.L. Gardner (FRS) - Sponsor.
1976/79	Special Postdoctoral Research Fellow (American Cancer Society -spf-18). Laboratory of Experimental Mammalian Embryology, Univ. of Oxford, Oxford - Professor R.L. Gardner, FRS.
1978/79	Research Fellow (NIH), Dept. of Zoology, Laboratory of Experimental Mammalian Embryology, Univ. of Oxford, Oxford. Professor R.L. Gardner (FRS) - Sponsor.
1979/80	Visiting Research Worker (NIH), Sir William Dunn School of Pathology, Univ. of Oxford, Oxford.
1979/80	Research Fellow (NIH), Botany School, Laboratory of Cytology, Univ. of Oxford, Oxford. Professor F. Whatley, FRS, and Dr C. Vesa (Sponsors).
1980/81	Honorary Senior Registrar, Neuropathology, The Radcliffe Infirmary, Oxford.
1980/81	Research Fellow (NIH), Dept. of Neuropathology, The Radcliffe Infirmary, Oxford.
1982	Senior Registrar, Neuropathology, The Radcliffe Infirmary, Oxford.
1982	Pathologist to H.M. Coroner, Mr N.G. Gardiner (Oxfordshire).
1983	(Locum) Consultant Neuropathologist, The Radcliffe Infirmary, Oxford June 3-17, 1983.
1984	Co-recipient, Health & Safety Executive project grant award: co-recipient with Professor P. Shubik, 'Iron Chelation: An attempt to develop a possible therapy for those at risk from exposure to asbestos by inhibiting the formation, persistence, and effects of asbestos bodies in vivo'.

MEMBERSHIPS

Oxford Uni- -sity Computer Centre Users Group	1985
Faculty of Biological Sciences, Univ. Oxford	1984
Subfaculty of Biochemistry, Univ. Oxford	1984
British Medical Association (BMA)	1984
Oxford Proton Microprobe Group, Dept. of Nuclear Physics, Univ. Oxford	1983
Neurosciences Specialty Group, Univ. Oxford	1981
British Society of Neuropathologists (BSN)	1981
Royal College of Pathology (Asso.)	1981
Neurofibromatosis Foundation (USA and UK)	1981
Tuberous Sclerosis Association (UK)	1981
British Society Development Biologists (BSDB)	1979
St Edmund Hall (Middle Common Room), Univ. Oxford	1976

AWARDS

Oxford District Research Grant Award (Embryonic Regulation of Neural Neoplasia)	Nov 1984	July 198-
Health & Safety Executive Project Grant Award (Potential Therapy... Asbestosis)	Sept 1984	July 1985
Medical Research Council Project Grant Award (Embryonic Regulation of Neural Neoplasia)	April 1982	Sept 1984
National Inst. Health Postdoctoral Training Award (Control of Trophoblastic Growth)	June 1978	Sept 1980
American Cancer Society Special Postdoctoral Award (Control of Trophoblastic Growth)	June 1976	June 1978
Intl. Agency for Research Cancer Postdoctoral Award (Control of Trophoblastic Growth)	Dec 1976	June 1977
Intl. Union Against Cancer (ICRETT) Award (Comparative Pulmonary Oncology)	June 1977	July 1978

RESEARCH EXPERIENCE

The applicant has maintained, as a persistent theme throughout his career, interests in both the basic and clinical aspects of the tumour problem. These are outlined below.

Rockefeller University, 1974-1975 - The applicant, in collaboration with Dr E.C.Diacumakos (Rockefeller University) and Dr W.B.Jones (Memorial Hospital for Cancer), was the first to grow choriocarcinoma *in vitro* without recourse to animal hosts, work conducted in a combined clinical and experimental study of the relationship between HCG secretion and malignant trophoblastic growth. The results of this study suggested that the secretory activity and cellular proliferation of malignant trophoblast may, at times, be dissociated phenomena *in vitro*, a conclusion confirmed by observations made, on rare occasions, *in vivo*. Since these experiments were originally aimed at mapping the HCG gene via cell hybridisation, the applicant also mastered techniques, developed by Dr Diacumakos, for microsurgical cell fusion, chromosome extraction, nuclear and cytoplasmic cell injection, and single cell cloning as well as methods needed for the cytogenetic analysis of such cloned cell lines.

Whilst at Rockefeller, the applicant also had numerous discussions with Professor Armin Braun on Epigenetic theory, tumour suppression, and recovery. These were based on Professor Braun's Crown Gall Teratoma Model System and laid the foundation for some of the applicant's future experiments.

Oxford University, 1976-1981 - Dept. Zoology - The perplexities of working with a malignant tissue that, even in its normal state, often behaved in a manner similar to a tumour, led the applicant to pursue studies of normal trophoblastic growth and developmental mechanisms generally thought to be related to neoplasia. Such studies were pursued in Oxford in the microsurgery laboratory of Prof. R.L.Gardner, a leading experimental mammalian embryologist. These studies culminated in a series of publications (described under 'Doctoral dissertation') and used a wide range of techniques including microsurgical injection, dissection, enzymatic separation, and/or tissue culture of pre- and post-implantation embryos and their constituent tissues. The latter were analysed biochemically, histochemically, cytologically, cytogenetically, histologically, and cytophotometrically using, in some cases, methods developed by the applicant. Embryo transfer and tumour inoculation methods were also employed in these studies.

Oxford University, 1980-1981 - Dept. of Botany - Preparations of trophoblastic giant cells were analysed cytologically, cytogenetically, and cytophotometrically in the Botany School with the advice of two eminent cytologists, Dr C.C.Vasa and Dr F.A.L.Clowes. These analyses were primarily directed at assessing DNA synthesis and chromosome structure and the results are described under 'Doctoral dissertation'.

Oxford University, 1980-1981 - Sir William Dunn School of Pathology - Embryonal carcinoma (EC) cell-derived mouse chimaeras were produced by the applicant using Prof. Gardner's blastocyst injection methods and the mature chimaeric tissues were subsequently dissected into their constituent parts, ectopically transferred to syngeneic hosts, and observed in host sites for tumour growth of donor cell origin. Since ectopically transferred, chimaeric, placental tissues were the only ones to form tumours, it was concluded that placental, in contrast to somatic tissues, lacked the ability to suppress the tumorous phenotype. Discussions with Prof. Armin Braun subsequently led the applicant to ask if tumour cell types other than those of EC cell origin, could colonise the embryo and, in turn, be suppressed or regulated by the embryonic environment. Since it was likely that this would occur with 'developmentally synchronised', donor-host cell combinations, e.g. when neuroblastomas were injected into the normal neuroblastic tissues of the embryo, a microsurgical method was developed for the manipulation of postimplantation mouse embryo brain *in utero*.

Oxford University, The Radcliffe Infirmary, 1981-1985 - Dept. of Neuropathology. This method was developed in the Dept. of Neuropathology, The Radcliffe Infirmary, Oxford. Although the method permitted injections to be performed with marker substances, cells were never injected. Still the materials needed to produce the appropriate donor-host cell combinations were obtained, these differing in β-Glucuronidase (-Gus) and Glucose

RESEARCH EXPERIENCE

The applicant has maintained, as a persistent theme throughout his career, interests in both the basic and clinical aspects of the tumour problem. These are outlined below.

Rockefeller University, 197- -75 - The applicant, in collaboration with Dr G.G.Diacumakos (Rockefeller University) and Dr W.B.Jones (Memorial Hospital for Cancer), was the first to grow choriocarcinoma *in vitro* without recourse to animal hosts, work conducted in a combined clinical and experimental study of the relationship between HCG secretion and malignant trophoblastic growth. The results of this study suggested that the secretory activity and cellular proliferation of malignant trophoblast may, at times, be dissociated phenomena *in vitro*, a conclusion confirmed by observations made, on rare occasions, *in vivo*. Since these experiments were originally aimed at mapping the HCG gene via cell hybridisation, the applicant also mastered techniques, developed by Dr Diacumakos, for microsurgical cell fusion, chromosome extraction, nuclear and cytoplasmic cell injection, and single cell cloning as well as methods needed for the cytogenetic analysis of such cloned cell lines.

Whilst at Rockefeller, the applicant also had numerous discussions with Professor Armin Braun on Epigenetic theory, tumour suppression, and recovery. These were based on Professor Braun's Crown Gall Teratoma Model System and laid the foundation for some of the applicant's future experiments.

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Oxford University, 1980-1981 - Dept. of Botany - Preparations of trophoblastic giant cells were analysed cytologically, cytogenetically, and cytophotometrically in the Botany School with the advice of two eminent cytologists, Dr C.C.Vasa and Dr F.A.L.Clowes. These analyses were primarily directed at assessing DNA synthesis and chromosome structure and the results are described under 'Doctoral dissertation'.

Oxford University, 1980-1981 - Sir William Dunn School of Pathology - Embryonal carcinoma (EC) cell-derived mouse chimaeras were produced by the applicant using Prof. Gardner's blastocyst injection methods and the mature chimaeric tissues were subsequently dissected into their constituent parts, ectopically transferred to syngeneic hosts, and observed in host sites for tumour growth of donor cell origin. Since ectopically transferred, chimaeric, placental tissues were the only ones to form tumours, it was concluded that placental, in contrast to somatic tissues, lacked the ability to suppress the tumorous phenotype. Discussions with Prof. Armin Braun subsequently led the applicant to ask if tumour cell types other than those of EC cell origin, could colonise the embryo and, in turn, be suppressed or regulated by the embryonic environment. Since it was likely that this would occur with 'developmentally synchronised', donor-host cell combinations, e.g. when neuroblastomas were injected into the normal neuroblastic tissues of the embryo, a microsurgical method was developed for the manipulation of postimplantation mouse embryo brain *in utero*.

Oxford University, The Radcliffe Infirmary, 1981-1985 - Dept. of Neuropathology. This method was developed in the Dept. of Neuropathology, The Radcliffe Infirmary, Oxford. Although the method permitted injections to be performed with marker substances, cells were never injected. Still the materials needed to produce the appropriate donor-host cell combinations were obtained, these differing in β-Glucuronidase (-Gus) and Glucose

DOCTORAL DISSERTATION - SUMMARY

1. Control of trophoblastic proliferation in early postimplantation mouse development:

Several conditions are needed to promote the growth of postimplantation mouse trophoblast. Firstly, an appropriate tissue shape and close cell contacts are needed to suppress the trophoblastic giant cell transformation and maintain these tissues in a largely diploid, non-giant state. Moreover, since an appropriate tissue shape cannot, on its own, promote trophoblastic cell division, ICM-derivatives, e.g. extraembryonic endoderm and/or mesoderm, are probably also needed to stimulate normal trophoblastic growth.

2. Control of trophoblastic proliferation in the guinea pig:

The growth of guinea pig trophectoderm and its early postimplantation derivatives appear to be under ICM control. Thus, the multilayered attachment cone of the guinea pig blastocyst is probably not due to an ICM-independent proliferation of trophectoderm but is most likely derived from a transient accumulation of abembryonic trophectodermal cells.

3. Origin of the trophoblastic giant cells: binucleation

In the mouse, postimplantation trophoblast appears to become giant through a binucleate phase. Thus, the growth of mouse trophectoderm occurs, to a certain extent, in a manner similar to the polyploidisation of mouse liver.

4. Origin of the trophoblastic giant cell: multinucleation

The finding of multinucleates in pure, trophectoderm-derived tissues suggests that trophoblastic multinucleation, at least in the mouse, does not depend upon prior contact with mesenchyme or mesenchymal tissues. Multipolar, polyploid mitotic figures were found in mouse decidua, tissues known to contain multinucleates similar to the ones seen in the trophoblastic derivatives of ruminants. This suggests that the multinucleate cells seen in some forms of trophoblast may arise in a manner similar to those found in rodent decidual tissues, i.e. via a multipolar mitosis without concomitant cytokinesis.

5. Mechanism by which DNA accumulates during the trophoblastic giant cell transformation

Polyploid metaphases were found within postimplantation mouse trophoblast that had chromosomes arranged either randomly or in pairs. This suggests that at least some mouse trophoblastic cells increase their DNA via polyploid mitotic and/or endomitotic cycles.

6. Control of the trophoblastic giant cell transformation

The degree to which trophectoderm-derived cells endoreduplicate their nuclear genome and thus become giant can be influenced by changes in tissue shape and the extent of intercellular contact. Thus, cell separation, spreading, and stretching appear to promote the giant cell change not only within trophectodermal derivatives but also within tissues known to behave in a manner similar to trophectoderm, namely extraembryonic endoderm. Moreover, the giant cells found in normal and neoplastic extraembryonic tissues are not necessarily terminal and incapable of cell division since they may be found in mitosis either before and/or following mitogenic stimulation. Comparative studies also suggest that the onset of the giant cell change in one extraembryonic membrane may be temporally correlated with the development and/or degeneration of a nearby placental tissue layer.

Phosphate Isomerase (GPI) alleles. The putative donor cells, Ependymoblastoma (Ep) were GPI-A - -Cus (heat stable) derived from A/J mice and the host embryos were GPI-B - -Cus (heat labile) derived from C57BL mice, the EP tumour being able to grow within and kill C57BL adults. Both tumour cell lines and mouse colonies were maintained for almost two years.

In addition to these experimental studies, several histopathological reviews were performed. The first of these described, in separate publications, unusual associations in hamartomatous dysgenetic disorders either of neural crest (e.g. Intra-cranial melanoma and Naevus of Ota) or non-neural crest (e.g. Tuberous Sclerosis with Sarcomas and Adenocarcinomas) origin. The second review provided the first evidence to suggest that the NF gene in Neurofibromatosis may not only predispose the neural crest-derived Schwann cells to undergo malignant change but also may affect, in a similar way, the non-neural crest, glial derivatives of the cerebellum. The applicant also analysed some of the largest series of ependymomas, cerebellar astrocytomas, medulloblastomas, and craniopharyngiomas using computer-assisted techniques and determined a variety of clinical-pathological prognostic indices for each of these tumour types.

Oxford University, 1984 - Dept. of Nuclear Physics - The applicant, in collaboration with the Oxford proton microprobe group, was the first to demonstrate the endogenous elements of the human brain with the proton microprobe thereby generating an 'elemental map' which closely corresponded to the normal architecture of that tissue.

Imperial Cancer Research Fund, Tumour Reference Collection (TRC) and the San Diego Zoological Research Hospital (SDZ), 1976 - The applicant made an extensive histopathological review of a large series of pulmonary tumours found in captive wild animals (SDZ) and various domestic species (TRC), the results confirming previous reports that most animal lung tumours are of the alveolar type, i.e. the one not generally seen in association with smoking.

Oxford University/Toxicology, 1985 to present - Under the direction and with the assistance of Prof. P.Shubik, the applicant has been investigating methods of modifying the inflammatory response to crocodilite and the zeolite, erionite. Such investigations are aimed at blocking the formation of the ferruginous body and the accumulation of excess tissue iron in the hope that this will lessen the inflammatory response and the attendant risk of neoplasia which follows the inflammation.

PUBLICATIONS - Articles

1. Ilgren, E.B. (1980). Polyploidization of extraembryonic tissues during mouse embryogenesis. J. Emb.exp.Morph. 59:103-111.
2. Ilgren, E.B. (1980). The control of trophoblastic growth in the guinea pig. J.Emb.exp.Morph. 60:405-418.
3. Ilgren, E.B. (1981). The control of trophoblastic growth in the mouse: homotypic cellular interactions and polyploidy. J.Emb.exp.Morph. 62:183-202.
4. Ilgren, E.B. and Littlefield, J.W. (1981). The extraembryonic endodermal differentiation and polyploidization of embryonal carcinoma cells *in vitro*. Differentiation 19:115-20.
5. Ilgren, E.B. (1981). The *in vitro* morphogenesis of the guinea-pig egg cylinder. Anatomy and Embryology 163:351-365.
6. Ilgren, E.B. (1981). Placental polyploidy: a comparative study. Placenta 2:333-342.
7. Ilgren, E.B. (1981). The control of trophoblastic growth in the mouse: binucleation and polyploidy. Placenta 2:317-332.
8. Ilgren, E.B., Griner, L., Benirschke, K. and Pang, L.S.C. (1981). A comparative study of pulmonary tumours. Pathology Annual 17:331-351.
9. Ilgren, E.B., Briggs, M. and Aynesley-Green (1982). Precocious puberty and ganglionic hamartomas of the parapituitary. Clin.Neuropath. 2:95-98.
10. Ilgren, E.B. and Vaux, D. (1982). Comparative morphological study of mouse and human teratoma. Cancer Surveys 2(1):210-215.
11. Ilgren, E.B. (1983). Review article: Control of trophoblastic growth. Placenta 4:307-328.
12. Ilgren, E.B., Evans, E.P. and Birtenshaw, M.D. (1983). Origin of the multinucleate decidual cell of the mouse. Chromosoma 88:313-322.
13. Ilgren, E.B. (1983). Cardiomyogenic differentiation and teratocarcinoma formation in EC-derived chimeric placentae. Placenta 4:415-422.
14. Ilgren, E.B., Stiller, C., Stackel, M., Silberman, P. and Hughes, J.T. (1983). Ependymomas - A serial study of their growth, Part I. Biological Features. Clin.Neuropath. 3:113-121.
15. Ilgren, E.B., Stiller, C., Stackel, M., Silberman, D., Hughes, J.T. and Kaye, A. (1983). Ependymomas - A serial study of their growth, Part II. Survival Features. Clin.Neuropath. 3:122-127.
16. Ilgren, E.B., Westmoreland, D. and Adams, C.B.T. (1983). Cerebellar Pseudotumour due to Candida (Cerebellar Candidoma). J.Neurosurg. 60:428-430.
17. Pearson, J., Ilgren, E.B. and Spriggs, A.J. (1983). Lymphoma cells in cerebrospinal fluid confirmed by chromosome analysis. J.Clin.Path. 35:1307-1311.
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PLF Ilgren
DEF EX 3
6/21/94 DMB

CURRICULUM VITAE

Dr E.B. Ilgren, MA (Oxon), MD., D.Phil.(Oxon), MACPath., MRCPath. (Associate/Neuropathology)

Date of Birth: 13.viii.50

Place of Birth: Philadelphia: Pennsylvania, USA.

Nationality: American

PRESENT POSITION

Member of Faculty of Biological and Agricultural Sciences
and Subfaculty of Biochemistry, University of Oxford

EDUCATION

INSTITUTE AND LOCATION	DEGREE	YEAR CONFERRED	FIELD OF STUDY
Hahnemann Medical University	MD	1974	Medicine
American College of Pathology	MACPath	1976	Anatomic Pathology
University of Oxford	D.Phil (Oxon)	1980	Zoology/Botany
Royal College of Pathology UK (Primary)	MRCPath	1982	Neuropathology
University of Oxford, Oxford, UK.	MA (Oxon)	1983	Biology/Medicine

RESEARCH AND/OR PROFESSIONAL EXPERIENCE - CHRONOLOGY

1973 Student Worker, Dept. of Comparative Neuroanatomy, The Bryn Mawr College, Bryn Mawr, Pennsylvania.

1974 Doctorate of Medicine, The Hahnemann Medical College, Philadelphia, Pennsylvania, USA.

1973-74 Visiting Resident, The Memorial Hospital for Cancer & Allied Diseases, attached to Dr W.S. Jones, Consultant Gynaecological Surgeon, Trophoblastic Disease Centre.

1974 Assistant Pathologist, The New York Hospital - Cornell University Medical Centre, New York City.

1975 Assistant Forensic Pathologist, Office of the Medical Examiner, The New York University Medical Centre, Dr M.C. Baden (Sponsor) - Chief Medical Examiner.

1975 Diplomate of the National Board of Medical Examiners (USA).

1975/76 Visiting Scientist, Rockefeller Institute of Medical Research, Professor A.C. Braun (Head of Department), Dr E.G. Diacumakos (Laboratory of Cytobiology, Head of Department).

1975 Licensed in Medicine and Surgery, State of New York, No. 124849.

1976 Member, American College of Pathologists, Board Certified, American Board of Pathology, Anatomic Pathology.

1976 Visiting Worker The Imperial Cancer Research Fund, Dept. of Pathology, London - Professor R.A. Willis and Dr L.S.C. Pang (Head of Department).

1977 Visiting Research Scientist (Univ. of South California at San Diego, San Diego Zoological Society and Dept. of Pathology, Professor K. Benirschke - Sponsor.

1977 Travelling Research Fellow (International Union Against Cancer) - Geneva, Switzerland.

1976/77 Postdoctoral Research Fellow (International Agency for Cancer Research/World Health Organisation) Dept. of Zoology, University of Oxford, Professor R.L. Gardner (FRS) - Sponsor.

1976/79 Special Postdoctoral Research Fellow (American Cancer Society - spf-14) Laboratory of Experiment Mammalian Embryology, Univ. of Oxford, Professor R.L. Gardner (FRS).

1978/79 Research Fellow (NIH), Dept. of Zoology, Laboratory of Experimental Mammalian Embryology, Univ. of Oxford, Professor R.L. Gardner (FRS) - Sponsor.

1979/80 Visiting Research Worker (NIH), Sir William Dunn School of Pathology, Univ. of Oxford, Oxford.

1979/80 Research Fellow (NIH), Botany School, Laboratory of Cytology, Univ. of Oxford, Oxford, Professor F. Whatley, FRS, and Dr C. Vasa (Sponsors).

1980 D. Phil. (Oxon), Dept. of Zoology & Botany, Univ. of Oxford, Member of St Edmund Hall.

1980/81 Honorary Senior Registrar, Neuropathology, The Radcliffe Infirmary, Oxford.

1980/81 Research Fellow (NIH), Dept. of Neuropathology, The Radcliffe Infirmary, Oxford.

1982 Senior Registrar, Neuropathology, The Radcliffe Infirmary, Oxford.

1982 Pathologist to H.M. Coroner, Mr N.G. Gardiner (Oxfordshire).

1982 General Medical Council, Full Registration.

1982 Recipient, MRC Project grant award 'Embryonic Regulation of Neural Neoplasia'

1983 MA (Oxon) - Status: Primary, MRCPath (Neuropathology).

1983 (Locum) Consultant Neuropathologist, The Radcliffe Infirmary, Oxford, June 3-17, 1983.

1983 Member, Department of Biochemistry, University of Oxford, Subfaculty.

1983 Member, Oxford Nuclear Physics Proton Microprobe Users Group.

1984 Member, Faculty of Biological Sciences, University of Oxford.

1984 Co-recipient, Health & Safety Executive project grant award: co-recipient with Professor P. Shubik "Iron Chelation: An attempt to develop a possible therapy for those at risk from exposure to asbe by inhibiting formation, persistence, and effects of asbestos bodies in vivo".

1985 Recipient, Oxford Area Health Authority/District Research Grant Award "Embryonic regulation of ne neoplasia.

... Consultant Neuropathologist, The Radcliffe Infirmary, Oxford, June 3-17, 1983.
1983 Member, Department of Biochemistry, University of Oxford. Subfaculty.
1983 Member, Oxford Nuclear Physics Proton Microprobe Users Group.
1984 Member, Faculty of Biological Sciences, University of Oxford.
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1985 Recipient. Oxford Area Health Authority/District Research Grant Award "Embryonic regulation of neu neoplasia".

Dates (month & year) From	To	Name and address of sponsor/ employer (block letters)	
June 1974	June 1976	New York Hospital/Cornell University Med. Centre	Assistant Pathologist, Anatomic Path. (Registrar/Sr. Reg. Grade)
June 1976	Sept. 1976	Office of the Medical Examiner (NY) New York Univ. Med. Centre	Assistant/Pathologist, Forensic Path. (Sr. Reg./Consultant Grade)
Dec.1976	June 1977	Intl. Agency for Research on Cancer/World Health Org. (Lyon)	Research Fellow, Embryology, Cell Biology, Oncology (also see 'Research Experience')
June 1976	June 1978	American Cancer Soc. (NY)	Research Fellow, Embryology, Cell Biology, Oncology
June 1978	Sept. 1980	NIH Bethesda, MD)	Research Fellow, Embryology, Cell Biology . Oncology
Sept. 1980	June 1985	HM Coroner (Oxford)	Forensic Neuropathology. (Senior Registrar/ Consultant (Locum) Grade)
Sept. 1980	June 1985	National Health Service (UK)	Neuropathologist (Senior Registrar/Consultant (Locum) Grade)
Dec. 1984	Dec. 1985	Health & Safety Executive (London)	Research Pathologist (see 'Research Experience')
July 1984	Apr. 1987	Univ. of Oxford, and	Toxicologist: Research Fellow: Faculty Member (Biological Sciences), Subfaculty (Biochemistry
		American Industrial Health Council (AIHC) International Life Sciences Institute (ILSI) Certified Color Manufacturers Association (CCMA) Flavor Extract Manufacturers Association (FEMA) Research Institute for Fragrance Materials (RIFM) Chemical Manufacturers Association (CMA) Procter & Gamble Pepsico: Coca-Cola Co. and DHSS, Dept. of Toxicology (London)	
		Imperial Cancer Research Fund, Clinical Trials Study Unit, Nuffield Dept. of Clinical Medicine, University of Oxford.	Research Officer (Hon.)
April 1989	Present	University of Oxford, St Edmund Hall	Member, Faculty (Biological Sciences) Subfaculty (Biochemistry)

MEMBERSHIPS

Oxford University Computer Centre Users Group	1985
Faculty of Biological Sciences, University of Oxford	1984
Subfaculty of Biochemistry, University of Oxford	1984
British Medical Association (BMA)	1984
Oxford Proton Microprobe Group, Dept. of Nuclear Physics, University of Oxford	1983
Neurosciences Speciality Group, University of Oxford	1981
British Society of Neuropathologists (BSN)	1981
Royal College of Pathology (Assoc.)	1981
Neurofibromatosis Foundation (USA and UK)	1981
Tuberous Sclerosis Association (UK)	1981
British Society Development Biologists (BSDB)	1979
St Edmund Hall (Middle Common Room), University of Oxford	1976

AWARDS

Oxford District Research Grant Award (Embryonic Regulation of Neural Neoplasia)	Nov. 1984	July 1985
Health & Safety Executive Project Grant Award (Potential Therapy... Asbestosis)	Sept. 1984	July 1985
Medical Research Council Project Grant Award (Embryonic Regulation of Neural Neoplasia)	Apr. 1982	Sept. 1984
National Inst. Health Postdoctoral Training Award (Control of Trophoblastic Growth)	June 1978	Sept. 1980
American Cancer Society Special Postdoctoral Award (Control of Trophoblastic Growth)	June 1976	June 1978
Intl. Agency for Cancer Research (WHO) Postdoctoral Award (Control of Trophoblastic Growth)	Dec. 1976	June 1977
Intl. Union Against Cancer (ICRETT) Award Comparative Pulmonary Oncology)	June 1977	July 1978

DOCTORAL DISSERTATION - SUMMARY

1. Control of trophoblastic proliferation in early postimplantation mouse development:

Several conditions are needed to promote the growth of postimplantation mouse trophoblast. Firstly, an appropriate tissue shape and close cell contacts are needed to suppress the trophoblastic giant cell transforma and maintain these tissues in a largely diploid, non-giant state. Moreover, since an appropriate tissue sh cannot, on its own, promote trophoblastic cell division, ICM-derivatives, e.g. extraembryonic endoderm and mesoderm, are probably also needed to stimulate normal trophoblastic growth.

2. Control of trophoblastic proliferation in the guinea pig:

The growth of guinea pig trophectoderm and its early postimplantation derivatives appear to be under ICM control. Thus, the multilayered attachment cone of the guinea pig blastocyst is probably not due to an-ICM independent proliferation of trophectoderm but is most likely derived from a transient accumulation of abembryonic trophectodermal cells.

3. Origin of the trophoblastic giant cells: binucleation

In the mouse, postimplantation trophoblast appears to become giant through a binucleate phase. Thus, the growth of mouse trophectoderm occurs, to a certain extent, in a manner similar to the polyploidisation of mouse liver.

4. Origin of the trophoblastic giant cell: multinucleation

The finding of multinucleates in pure, trophectoderm-derived tissues suggests that trophoblastic multi-nucleation, at least in the mouse, does not depend upon prior contact with mesenchyme or mesenchymal tissue Multipolar, polyploid mitotic figures were found in mouse decidua, tissues known to contain multinucleates similar to the ones seen in the trophoblastic derivatives of ruminants. This suggests that the multinucleat cells seen in some forms of trophoblast may arise in a manner similar to those found in rodent decidual tissues, i.e. via a multipolar mitosis without concomitant cytokinesis.

5. Mechanism by which DNA accumulates during the trophoblastic giant cell transformation

Polyploid metaphases were found within postimplantation mouse trophoblast that had chromosomes arranged either randomly or in pairs. This suggests that at least some mouse trophoblastic cells increase their DNA via polyploid mitotic and/or endomitotic cycles.

6. Control of the trophoblastic giant cell transformation

The degree to which trophectoderm-derived cells endoreduplicate their nuclear genome and thus become giant can be influenced by changes in tissue shape and the extent of intercellular contact. Thus, cell separation spreading, and stretching appear to promote the giant cell change not only within trophectodermal derivativ but also within tissues known to behave in a manner similar to trophectoderm, namely extraembryonic endoder Moreover, the giant cells found in normal and neoplastic extraembryonic tissues are not necessarily termina and incapable of cell division since they may be found in mitosis either before and/or following mitogenic stimulation. Comparative studies also suggest that the onset of the giant cell change in one extraembryonic membrane may be temporally correlated with the development and/or degeneration of a nearby placental tissue layer.

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2. Ilgren, E.B. (1980). The control of trophoblastic growth in the guinea pig. J. Emb. exp. Morph. 60
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4. Ilgren, E.B. and Littlefield, J.W. (1981). The extraembryonic endodermal differentiation and polyploidization of embryonal carcinoma cells in vitro. Differentiation 19:115-20.
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9. Ilgren, E.B., Briggs, M. and Aynesley-Green (1982). Precocious puberty and ganglionic hamartomas of parasituitary. Clin. Neuropath. 2:95-98.
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12. Ilgren, E.B., Evans, E.P. and Sirtenshaw, M.D. (1983). Origin of the multinucleate decidual cell of mouse. Chromosoma 48:313-322.
13. Ilgren, E.B. (1983). Cardiomyogenic differentiation and teratocarcinoma formation in EC-derived chim placentae. Placenta 4:415-422
14. Ilgren, E.B., Stiller, C., Steckel, M., Silberman, P. and Hughes, J.T. (1983). Ependymomas - A serial study of their growth. Part I. Biological Features. Clin. Neuropath. 3:115-121.
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CONSULTANCIES

Cola Cola (1983)	Position paper: Aspartame and brain function
PEC (Panel on Caffeine Safety) Zurich (1985-1986)	Caffeine Literature 'safety' assessment
HSC (Holland Sweetener Co.) Maastricht (1986-Present)	Aspartame Literature 'safety' assessment
UniRoyal: Technical Assessment Systems (TAS): McKenna, Conners, Cuneo (Wash.)	Alar safety assessment
Armstrong World Industries/ Celotex (Asbestos)	Montgomery, McCracken, Walker, Rhoads (Phila.) Bloom and Green (Cinn., Ohio)
Basic Tile (Asbestos)	Arter & Hadden (Cleveland, Ohio) Budd, Larner, Gross (Newark, N.J.)
GAF Corporation (Asbestos)	Sherman & Picaddio (Pittsburgh, Pa.)
R.W. Johnson	Retinoid Safety Assessment

INVITED LECTURES

British Neuropathology Society (London) (Clinical and Prognostic Indices for Human Ependymomas)	1982
Ludwig Institute for Cancer Research, University of Bern (Regulation of Neural Neoplasia)	1983
Faculty of Medicine, University of Lausanne (Regulation of Neural Neoplasia)	1984
Institute for Brain Research, Zurich (Elemental of the Human Brain using the Proton Microprobe)	1984
Swiss Neuropathology Society (St Moritz) (Chronic Inflammation and Involvement of the Nucleus Ambiguus in MultiSystem Atrophy)	1985
Sloan Lecture. Bryn Mawr College.	1989

made this eighth day of October 1987
between the Oxford Publisher of the OXFORD UNIVERSITY PRESS of Walton Street Oxford OX2 6D: hereinafter called 'the Publisher' of the one part and Dr E.B. Ilgren, 13 Frenchay Road, Oxford: Dr P. Shubik, 13 Norham Gardens, Oxford.

hereinafter called 'the Author' of the other part

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Dr. D. H. Hughes
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June 20, 1986

Ms. Sharon Senzik
International Life Sciences Institute
1126 Sixteenth Street, N.W., Suite 111
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Subject: Promotion Project at Oxford University

Dear Sharon:

I want to thank you for your Association's participation in the support of the carcinogenesis promotion review project being conducted by Dr. Ed Ilgren at Oxford University under the supervision of Dr. Phil Shubik. The complete list of groups that are providing financial support is:

1. American Industrial Health Council (AIHC) - J. David Sandler
2. International Life Sciences Institute (ILSI) - Ms. Sharon Senzik
3. Certified Color Manufacturers Association (CCMA) - Dr. James Noonan
4. Flavor Extract Manufacturers Association (FEMA) - Dr. Richard L. Hall
5. Research Institute for Fragrance Materials (RIFM) - Dr. Richard A. Ford
6. Chemical Manufacturers Association (CMA) - Mr. Carl Umland
7. Procter & Gamble - Dr. Donald H. Hughes
8. Coca-Cola Company - Dr. James Emerson
9. Pepsico - Dr. James W. Stanley

As you may recall, Dr. Ilgren actually began work on this project in the fall of 1985 with tacit support from the National Cancer Institute. However, that support disappeared with the emergence of Gramm-Rudman. Hence, we approached you for help and your positive response was critical to the continuation of this project. We anticipate this association and industry support will carry Dr. Ilgren through October of 1986 and complete this phase of the project, which is to review and critique the massive literature on promotion and to have a paper ready for publication and presentation at an appropriate meeting at the end of this year. I plan to get back to you later in the summer with a status report and thoughts for the future of this project.

Very best regards.

Very truly yours,

Don

D. H. Hughes, Ph.D.
For the Science Committee

DHH/es

cc: Dr. Philippe Shubik, Dr. Edward Ilgren, Dr. W. Gary Flamm, Dr. Ian Munro, Dr. Robert J. Moolenaar, Dr. Richard H. Adamson, Mr. J. David Sandler

UNIVERSITY OF OXFORD

No. GSF/3A

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Oxford OX1 2JD
Telephone no.
(0865) 50747

DATE 31 January 1984

FACULTY MEMBERSHIP

The following name has been added to the list of members of the faculty indicated below under the provisions of the Statutes, Title VI, Sect. I, cl. 2 ( ). Communications concerning the faculty will be sent to the address given unless the Secretary of Faculties is informed otherwise.

Dr. Edward B. Ilgren, M.A. status, D.Phil., — Name

Department of Neuropathology, — Address
Radcliffe Infirmary,
Woodstock Road.

St. Edmund Hall — College

Biological and Agricultural Sciences — Faculty

Biochemistry — Sub-Faculty

UNIVERSITY OF OXFORD

UNIVERSITY OFFICES
WELLINGTON SQUARE
OXFORD
OX1 2JD

Personal Telephone: 0 865 270190

From the Head Clerk
25 April 1994

Ref. No. GEM/S

Dear Sir or Madam

I have received your letter of 21 April about Dr. K.B. Ilgren. He was a member of the University's staff from 1984 to 1987 when he left; I do not have a forwarding address.

He was a member of St Edmund Hall, within the Mathematics, I suggest ... contact the college office for possible further information. The fax number is 0865 279090.

Yours faithfully

Philip Moss

P.W. Moss

Lynnae T. Waskosky
Legal Assistant
Stich, Angell, Kreidler & Muth, PA,
The Crossings, Suite 120
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PLF Ilgren
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TOTAL P.02

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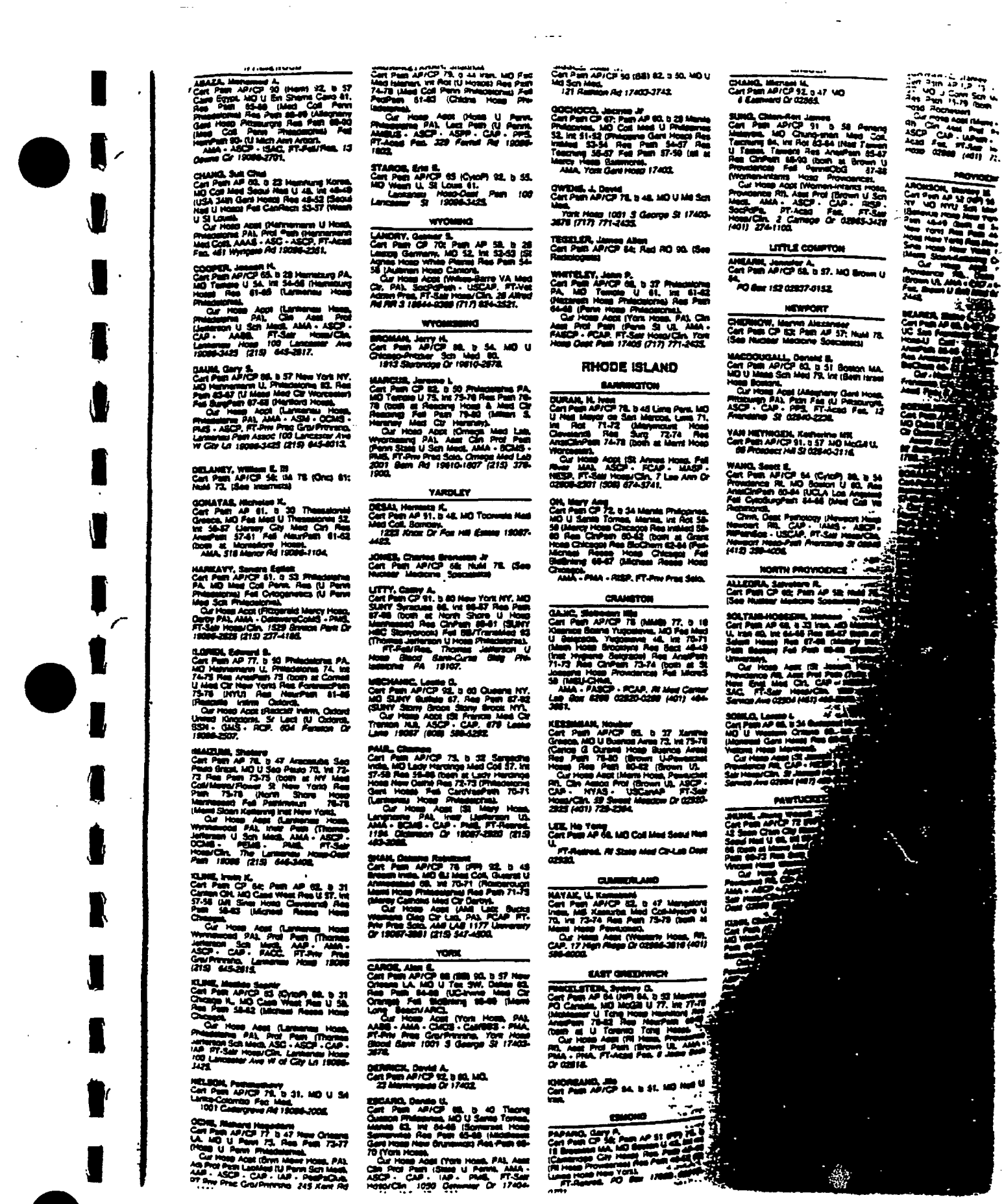

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Ilgren 6
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6/11/94 LMB

FACSIMILE FRONT SHEET

ATTENTION OF:...MS LYNNAE I. WASKOSKY.....PAGE 1 OF...1.....

INSTITUTION/DEPT....STITCH, ANGELL, KREIDLER DATE:...28.4.94...

ADDRESS:...AND MUTH, P.A....250 SECOND AVENUE TIME:...14.10.....

SOUTH, SUITE 120, MINNEAPOLIS, MN 55401, ...REF:.....JCBG/SD
USA.

FAX NO.:....010-612-333-1940.....

SENDER: Mr J.C.B. Gosling, Principal

MESSAGE

Dear Ms Waskosky,

Dr E.B. Ilgren did his doctoral studies at this College. Later he had appointments at the Department of Neuropathology, Radcliffe Infirmary Hospital in Oxford, and was a member of the Faculty of Biological Sciences and sub-faculty of Biochemistry.

The situation at Oxford is somewhat complex, but Dr Ilgren was never actually on the staff of this College, although he was a member of the College in virtue of having been a graduate student here.

Yours sincerely,

Justin Gosling

J.C.B. Gosling,
Principal.

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ADDRESS:...AND.MUTH,.P.A....250.SECOND.AVENUE.TIME:..14.10.....

.SOUTH,.SUITE.120,.MINNEAPOLIS,.MN.55401,....REF:.....JCBG/SD..
USA.

FAX NO.:....010-612-333-1940.....

SENDER: Mr J.C.B. Gosling, Principal

MESSAGE

Dear Ms Waskosky,

Dr E.B. Ilgren did his doctoral studies at this College. Later he had appointments at the Department of Neuropathology, Radcliffe Infirmary Hospital in Oxford, and was a member of the Faculty of Biological Sciences and sub-faculty of Biochemistry.

The situation at Oxford is somewhat complex, but Dr Ilgren was never actually on the staff of this College, although he was a member of the College in virtue of having been a graduate student here.

Yours sincerely,

J.C.B. Gosling,
Principal.

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PLF Ilgren
DEF ex 2
6/21/94 D.M.B.

28 April 1994

Dear Ms Waskosky

Conrad v Union Carbide
Your file No: 13574

I am responding to your letter of 21 April received by fax.

There are two routes to membership (MRCPath) of this College: by examination (in two parts) or by submission of published work. Under the old examination system which is being phased out this year, it was also possible to gain exemption from the first part of the membership examination on the basis of an approved postgraduate qualification or submission of publications.

According to our records, Dr [redacted] B Ilgren of Apt 503, 830 Montgomery Avenue, Bryn Mawr, Pennsylvania 19010 is an Associate of the College, having gained exemption in October 1981 from the first part of the membership examination, the Primary, on the basis of his publications in a pathological field. The status of Associate is entirely optional on the part of the applicant: it does not permit entitlement to designated initials such as ARCPath but does allow the holder to receive the quarterly mailings of the College.

I hope this information is of help.

Yours sincerely

C Roberts.

Professor C Roberts
Registrar

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071-930 5863 (Sx. Meetings/Manpower)
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Sheehan, William W., MD
Strange, Jimmy R., MD
Taylor, James Richard, MD
White, Robert S., MD
Williams, Gregory P., MD

OREGON

Albany
Crow, Kenneth A., MD

Ashland
Jarvis, Thomas B., MD

Banks
Cochran, Terence H., MD

Beaverton
Dunn, Robert C., MD
Hurly, James M., MD
Hutchens, Tyra T., MD

Bend
Howbert, James P., MD
Judd, James L., MD
Mcgeary, George D., MD
Schneider, Roger A., MD
Yocom, Laurel B., MD

Clackamas
Holahan, Kathleen P., MD

Coos Bay
Hosack, William D., MD
McIntyre, Henderson M., MD, PhD

Corvallis
Apter, Roy J., MD
Lang, John L., MD
Lund, Paul K., MD
Phillips, Wayne H., MD
Vrtiska, Frank L., MD

Eugene
Fish, Mathews B., MD
Hales, Michael J., MD
Holmes, Robert O., MD
Houck, Jeffrey A., MD
Kahn, Brent D., MD
Liu, Curtis Roy, MD
Meyers, David S., MD
Miller, Jacqueline D., MD
Rutz, David A., MD
Starr, Grier Forsythe, MD
Vickers, L. Samuel, MD

Grants Pass
Hong, Fang-Yen, MD
Olson, James N., MD

Hillsboro
Pickert, Ted C., MD

Klamath Falls
Edwards, Robert N., I, MD

La Grande
Helou, Khalil Y., MD

Lake Oswego
Orfanakos, Nicholas G., MD
Oyama, Albert A., MD
Skibba, Charles T., MD

Medford
Buck, Robert H., MD
Newland, Gary L., MD
Stephenson, Robert Wayne, MD
Treger, Thomas R., MD
Watson, Frank H., MD

Milwaukie
Berroth, Margaret E., MD

Newberg
Connell, Robert M., MD

Ontario
Meier, Robert H., MD

Oregon City
Daut, Dennis P., MD

Pendleton
Adams, Lawrence J., MD
Clausen, SallyAnn G., MD
Sawyer, James B., MD

Portland
Aljazi, Shadab N., MD
Arntson, Eric W., MD
Baer, Daniel M., MD
Belsey, Richard E., MD
Boudousquie, Alan C., MD
Cannon, Scott L., MD
Cook, R. Edward, MD
Curl, Franklin D., MD
DeYoung, Patricia A., MD
Fandel, Teresa M., MD
Franzini, Daisy A., MD
Fuchs, Peter C., MD
Galey, William T., MD
Gonzalez-Vitale, Juan C., MD
Hammer, Elisabeth P., MD
Harris, Homer H., MD
Harry, Raymond Charles, MD
Harter, Jeffrey D., MD
Henschel, Eugene V., Jr., MD
Houghton, Donald C., MD
Johnson, David S., MD
Joseph, Edward, DO
Juengel, Randal Carl, MD
Klessling, Peter J., MD
Klint, William F., Jr. MD
Krum, Robert J., MD
Land, Rachiel N., MD
Landreth, Eugene W., MD
Longo, Julia C., MD
Marquardt, Victor C., Jr., MD
McDonald, Clark E., MD
Mehren, Laura Sue, MD
Miles, Catherine M., MD
Millas, Joan C., MD
Moore, Michael A., MD
Nixon, Randal R., MD, PhD
Odell, J. Michael, MD
Orr, James E., MD PhD
Owings, Raymond M., MD
Oyama, Kevin K., MD
Parsons, Catherine S., MD
Pickering, Nigel A., MD
Ray, Judith A., MD
Rosenheim, Sidney H., MD
Schafer, Donald W.E., MD
Schmidt, Waldemar A., MD, PhD
Shizaro, Steven L., MD
Shilling, Joel M., MD
Slavig, Richard E., MD
Smith, James M., MD
Spackman, Kent Alan, MD,PhD
Specht, H. David, MD
Thompson, Steven Keith, MD
Thorpe, John O., MD
Vennes, George J., MD
Wirt, Daniel Ptolemy, MD

Roseburg
Barnes, James L., MD
Ready, Michael T., MD

Salem
Beal, Melissa Lou, MD
Davis, Stanley K., MD
Jensen, Robert D., MD
Landers, Roger Q., III, MD
Lium, James H., MD
McKillop, Donald B., MD
Patton, Roy B., MD
Pozar, John M., MD
Rueckel, Douglas N., MD
VanPeenen, Hubert J., MD

Talent
Loquvam, George S., MD

The Dalles
Schubert, Fred C., Jr., MD

Tualatin
Seaman, Richard M., MD
Wallin, John L., MD

Warrenton
Nicholson, George R., MD

West Linn
Gordon, Melissa, MD

Wilsonville
Peterson, David A., MD

PENNSYLVANIA

Abington
Auerbach, Herbert E., DO
Chersky, Paul J., MD
Goldhahn, Richard T., Jr., MD
Huck, George F., Jr., MD
Klotz Jr., Roy G., MD

Allentown
Burkhard, Edward J., Jr., MD

Instructors for LAP printed in bold type.

Allentown (cont.)
Honeckman, Charles, DO
Klass, Athanasius C., MD
Kostanik, Frances V., MD
Little, Brian W., MD, PhD
Scott, Ralph H., MD

Allison Park
Cruz, Pedro A., MD
Dekker, Andrew, MD
Edwards, Rosemary, MD
Specht, Charles S., MD

Altoona
Jourdain, Luis M., MD
Kirsch, William J., MD
Snell, Eugene M., MD
Stanfield, Barbara L., MD

Aliquippa
Sheps, Earl S., MD

Ambler
Hagarty, John J., MD

Annville
Carmany, Thomas S., MD

Ardmore
Carroll, Deborah J., MD
Smith, M. Sue, MD
Wasik, Mariusz A., MD

Aspinwall
Finkelstein, Sydney D., MD

Audubon
Nikitas, Donald A., MD

Bala Cynwyd
Sun, Yongling, MD
Dotson, David James, MD
Dotson, Lynn H., MD

Bala-Cynwyd
Forest, Jean L., MD
Meyers, Karl R., MD
Uri, Antonia K., MD
Yarow, Noemi Susana, MD

Beaver
Saglio, Corrado M., MD
Marcus, Gary J., MD
Min, Tae C., MD
Reitz, John O., MD

Berwick
Hunter, Robert Gray, MD

Berwyn
Schramm, Harry A., MD

Bethel Park
Pradhan, Sulochana L., MD
Schubert, Eric D., MD

Bethlehem
Benz, Edward J., MD
Chiadis, James M., MD
Fisher, Joseph W., Jr., MD
Gonzalez, Rafael O., MD
Isaac, Leon A., MD
Longo, Santo, MD
Luft, Ronald D., MD
Lukaszczyk, Thomas A., MD
Mihalakis, Isidore, MD
Oliver, Jorge M., MD

Bloomsburg
Donon, R. Patrick, MD
Martin, John C., MD
Tilman, William E., MD

Blue Bell
Lieberman, Jill Mazzei, MD

Boothwyn
Campbell, William N., MD

Bowmansville
Lane, C. Darrell, MD

Braddock
Castro, Augusto D., MD
Kritcher, Charles, MD

Bristol
Song, Sang Won, MD

Brookhaven
Wilson, Roma A., MD

Brookville
Haverty, Gary F., DO

Brossmall
Daum, Gary S., MD

Bryn Mawr
Hausman, David H., MD
Hemert, Samia R., MD
Kasbegelan, Albert A., MD, PhD
Ledoslis, Charles T., MD
Miguel, Nemesio L., Jr., MD
Pietra, Giuseppe G., MD
Strumia, Paul V., MD
Williams, James R., MD

Butler
Kim, Yang K., MD
Moon, Sanggu, MD
Pirrello, Anthony M., Jr., MD

Camp Hill
Al-Annouf, Nabil, MD
Jenkins, Rosemary, MD
Maas, Anthony E., MD
Meniglia, Rosario, MD
Potok, Julian W., DO

Campbell
Bhattacharji, Susanta K., MD

Canonsburg
Syed, Tasneem A.Z., MD

Carlisle
Chang, Duck-Kyu, MD
Crist, Henry S., MD
Smith, James M., MD

Cedar Run
Corson, Margaret L., MD

Chadds Ford
Meier, Frederick A., MD

Chalfont
Aegerter, Ernest E., MD

Chalk Hill
Ayres, William W., MD

Chambersburg
Flanagan, Margaret M., MD
Hoffman, Howard L., MD
Ramirez, Constancio A., MD

Chester
Balin, Arthur K., MD, PhD
Lewis, James, DO
Spector, Harvey S., MD

Clarion
Puckett, James L., DO

Clarks Summit
O'Connor, James J., Jr., MD
Ross, Gary W., MD

Clearfield
Kennard, John F., MD
Reed, Michael F., MD

Coatesville
Casas, Angelina W., MD

Columbia
Sator, Susan M., MD

Conshohocken
Foldes, Julius, MD

Cooperstown
Lesko, Linda S., MD

Courtdale
Hudock, George E., Jr., MD

Dallas
Kashi, C. Warren, Jr., MD
Michalak, William A., MD
Wilde, William L., MD

Danville
Brett, David A., DO
Cera, Peter J., Jr., MD
Fevino, C. James, MD
Garbes, Archimedes D., MD
Hamati, Husam F., MD
Moran, John J., MD
Pascucci, Mary F., DO
Pierce, Richard N.
Rigotti, Richard M., MD
Schuerch, Conrad, MD
Wadih, George E., MD

Darby
Zotwaka, Mateka, MD

Devon
Moreno, Carlos, MD
Szymanski, Mark S., MD

Doylestown
Hawksley, Vaughn C., MD

Dresher
Schwartz, Heinz G., MD

Drexel Hill
Choi, John K., MD, PhD
Matthews, Lawrence M., Jr., MD
Monte, Steven A., MD

Du Bois
Costa, Jose M.L., MD

DuBois
Suslow, Gregory R., MD

East Stroudsburg
Cerra, Carmine J., MD
Gruszka, Frances A., MD
Tinsley, John P., MD

Easton
Altman, Arthur A., MD
Barnett, Charles P., MD
Brown, John M., MD
Dorman, Sandy A., MD
Fonseca, Carlos A., MD
Gaulin, J. Claude, MD
Harada, William A., MD
Salcedo, Wilfredo G., MD

Elkwood City
Williams, Karl E., MD

Elysburg
Reuter, Robert K., DO

Emmaus
Matalewicz, Theodore J., MD

Ephrata
Cota, Peter Christopher, MD

Erie
Eisenberg, Richard B., MD
Franklin, Norman, MD
German, Antonio L., MD
Jargess, Kenneth H., MD
Khera, Ritu, MD
Kiawon, David L., MD
Lara, Henry R.C., MD
Reitz, Mary E., MD
Rezwadewski, Jack V., MD
Ziegler, Thomas R., MD

Export
Talamo, Thomas S., MD

Farrell
Wilson, Steven Henry, MD

Fort Washington
Catalano, Edison, MD
Smith, Roberta Elaine, DO

Franklin
Davis, Bridgett K., MD
Sheppard, John W., MD
Suk, Jin H., MD

Gettysburg
Crippen, John W., MD
Mondelair Magdalena O., MD

Registrars for LAP printed in bold type.

PENNSYLVANIA

Gibsonia
Nathan, Girija S., MD
Payan, Husnang M., MD

Gladwyne
Katz, Sheila M., MD
Rango, Rena S., MD
Teufel, Severn, MD

Gladwynne
Pearlman, Eugene S., MD

Glenshaw
Hanrahan, James S., MD
Whiteside, Virginia E., MD

Glenside
Valenti, Salvatore M., MD

Greensburg
Beyer, Francis D., MD
Choi, Charles J., MD
Jetter, Walter W., MD
Morrow, Thayer K., MD
Ramos, Clarita P., MD

Greenville
Hawkins, Roger A., MD
Sobus, Peter C., MD

Gwynedd Valley
Balasubramanian, Manjula, MD

Hanover
Ehrhart, Kenneth W., MD
Socrates, Jess U., MD

Harrisburg
Bear, Robert S., DO
Berkheiser, S. W., MD
DiSanto, Susan K., MD
Gabriel, Hoda F., MD
Hartman, Ricky E., DO
Juveiler, Bernard W., MD
Olmstead, P. Michael, MD
Piper, James A., MD
Rady, Frank Raymond, MD

Hatboro
Siegel, Donald L., MD, PhD

Haverford
Ainsworth, Ann M., MD
Skiltarelic, Benedict, MD

Havertown
Mayer, Theodore G., MD
Stein, Donald B., Jr., MD

Hazleton
Koenig, Johann A., MD

Hermitage
Raisch, Frederick J., MD

Hershey
Abendroth, Catherine S., MD
Abt, Arthur B., MD
Berne, Cheryl A., MD
Frauenhoffer, Elizabeth E., MD
Granko, Ronald Trent, MD
Krieg, Arthur F., MD
Nasya, R. L., MD
Salam, Marquerite M., MD
Stortz, Cindy L., MD
Sturtz, Kraig W., MD
Ward, Samuel P., MD
Zaino, Richard J., MD

Holland
Gill, Manjit K., MD

Hollidaysburg
Anton, Americo B., MD
Cottle, Harold R., MD
Kurian, Santhamma, MD
Shaub, Howard G., Jr., MD

Homestead
Baker, Evan E., MD

Honesdale
Lee, Young W., MD

Horsham
Kotwal, Elchi K., MD

Hummelstown
Gramatovici, Mirela, MD

Huntingdon
Maytock, John H., MD

Huntingdon Valley
Horstmann, Joseph P., MD

Indiana
Dixon, Harold M., MD
Griffin, Steven P., MD
Martinez-Eskenasy, German, MD

Jeannette
DeCicco, Frank A., MD
Harster, Gerald Alfred, MD

Jenkintown
Griffith, Charles Q., MD

Johnstown
Benshoff, Albert M., MD
Bosis, Stephen T., MD
Evans, Hilary, MD
Goldblatt, Sidney A., MD
Montalecne, Paul N., MD
Ortiz, Roberto, MD
Patel, Sureshchandra A., MD
Pisano, James J., MD
Rizkalla, Waheeb M., MD
Vydareil, Mark R., MD
Yousef, Michael M., MD

King Of Prussia
Han, Aaron C., MD, PhD

King of Prussia
Schmidt, H. William, MD

Kingston
Rogers, Robert Allen, MD

Kittanning
Childs, James E., MD
Gerstbrein, Harry L., MD
Salinas, Joe A., MD

Lafayette
Swami, Vanita K., MD

Lancaster
Anthony, Christopher H., MD
Eastman, James T., III, MD
Eisenhower, Edward A., MD
Fabs, Gerald R., MD
Gerhard, Glenn Stephen, MD
Kryniak, Minerva P., MD
Nemoff, John, III, MD
O'Donnell, Ward M., MD
Penades, Enrique, MD
Regas, Mark C., MD, PhD
Umiker, William Oliver, MD
Young, J. Michael, MD

Lang Horne
Reyes, Jose M., MD

Langhorn
Ogden, Patrick G., DO

Langhorne
Ahmad, Rashat, MD
Frauenhoffer, Christopher, MD
Gibas, Zenon, MD, PhD
Souillard, Donald H., MD
Ziwer, Richard E., MD

Lansdale
Hansell, John R., MD
So, Andrew Lac, MD
Wright, David M., MD

Lansdowne
Belair, Patricia A., MD

Latrobe
Berardi, Ronald S., MD
Bures, Joseph Conrad, MD
Singh, Usha D., MD

Lebanon
Provencio, Florencio, MD
Sees, Jack N., Jr.,MD
Tanner, Leonard M., MD

Lewisberry
Bentz, Michael S., MD

Lewisburg
Lamparter, Robert W., MD
McTighe, Arthur H., MD

Lewistown
Smocks, Mark E., MD
Trivedi, Gopalkrishna M., MD

Ligonier
Gordon, Harold Eliot, MD

Lititz
Gaynor, William B., MD

Mechanicsburg
Tchang, Felix K.M., MD

Mackeyville
English, Walter E., Jr., MD

Malvern
Lisinger, John R., MD

Maple Glen
Khanna, Chanchal, MD

Mc Kees Rocks
Scheinman, Harold Z., MD

Mc Keesport
Past, Floranta P., MD

Meadowbrook
Fischer, Sandra L., MD
Rizzo, Thomas A., Jr., MD

Meadville
Eidbo, Joel R., MD
Schroeckenstein, Richard F., MD

Mechanicsburg
Kwee, Kim G., MD
Swamidoss, Stephenson S.P., MD

Media
England, James M., MD
Feldman, Michael D., MD, PhD
O'Hara, Brian J., MD
Patel, Prabha H., MD
Stevenson, Alan J., MD

Merion Station
Adler, Margaret J.S., MD
Hotman, Walter L, MD
Honigman, Frederic Herbert, MD
Lewis, Paul L., MD

Middletown
McGary, Carl T., MD, PhD
Meece, Christine A., MD

Mohnton
Welsh, James F., MD

Monroeville
Alhakim, Nawar M., MD
Bedetti, Carlos D., MD
Mirzabegi, Nematollah, MD
Myerowitz, Richard L., MD
Sharma, Kusum, MD
Viswanathan, Uma P., MD

Montoursville
Ahmed, Galal M., MD
Blom, Soren M. Ferrari, MD

Morgantown
Zaman, Syed Saeed, MD

Mount Penn
ChenSee, Jasper G., MD

Mount Pleasant
Watkins, Gloria J., MD

Mountaintop
Schmidt, Arnold P., MD

Narberth
Lease, James Arthur, DO
Paulson, James A., MD

Inspectors for LAP printed in bold type.

Natrona Heights
Bautista, Prime V., MD
Kim, Wha Sun, MD
Liang, Ping-Tchang, MD
Oehrle, John S., MD

Natrona Hts.
Miller, S.J.C., MD

New Castle
Fenner, H. E., MD
Isidro, Eugene G., MD
Ong, Bienvenido S., MD
Stoner, J. Fred, MD

New Cumberland
Murray, Cheryl Lynn, MD

New Hope
Kenny-Slizewski, Rose Mary, MD

New Kensington
Ahuja, Subash C., MD
Klieger, Herman L., MD
Ravano, Perumanan O., MD

Newton
Siderits, Richard H., MD

Newtown Square
Beasley, Eugene A., Jr., MD
Bhatt, Anjali G., MD
McGraw, John J., Jr., MD
Norris, Robert F., MD
Park, Hydow, MD
Sohn, Min, MD

Norristown
Belser, Paul Harper, MD
Bergnes, Manuel A., MD
Edelman, Andrew S., MD, PhD
Froio, Gregory F., MD
Gamarra, Violeta R., MD
Gupta, Chitra L., MD
Kashatus, William Charles, MD
McClements, Mary Jane, MD
McGinniss, Kathleen W., MD
Peale, Augustin R., MD
Tamaki, Hitoshi T., MD

Oaksdale
DiMarzio, Dante, DO

Oil City
Haji-Ojafari, Azizeh, MD

Palmyra
Phillips, Peter P., Jr., MD

Penn Valley
Bakhtar, Maryam, MD
Japko, Leonard I., MD

Philadelphia
Adas, Amy Ann, MD
Adesina, Adekunle M., MD
Al-Saleem, Tahseen Isa, MD
Alexander, Nancy J., MD
Alexandrin, Eugene, MD
Altman, Howard S., MD
Atkinson, Barbara F., MD
Baloch, Zubair W., MD
Bellin, Harvey J., MD
Bernard, David W., MD, PhD
Beydoun, Rafic, MD
Bhagat, Pradeep K., MD
Blodgett, Laura L., MD
Blondi, Robert J., DO
Blanchard, Cecily Chase, MD
Borer, William Z., MD
Brennan, John P., MD
Burrows, Stanley S., MD
Can, Seyit Adil, MD
Carpentieri, David F., MD
Cawley, Noel S., MD
Chaubal, Abhijeet V., MD
Ciccone, Emmet F., MD
Cohen, Stanley, MD
Conn, Rex B., MD
Decker, John P., MD
Dookhan, Dianne S., MD
Dowdy, Yvonne G., MD
Drapanski, John F., MD
Edmonds, Pamela R., MD
Edwards, Robin H., MD
Ehya, Hormoz, MD
Eulau, Stephen M., MD
Farano, Peter J., MD
Felgar, Raymond E., MD, PhD
Fernandes, Blanche L., MD
Fisk, Gordon S., MD
Fintel, Gerald C., MD
Fitz, Franklin, MD
Fitzpatrick, Brendan T., MD
Fox, Rosanne Mane, MD
Franchino, Charles J., MD
Gaia, Isidra H., MD
Gannon, Francis H., MD
Gatalica, Zoran, MD
Gotsdiner, Denise S., MD
Gupta, Prabodh K., MD
Haber, Marian Markowitz, MD
Hanau, Cheryl A., MD
Hasegawa, Michael W., MD
Hermann, George A., MD
Hood, Ian C., MD
Horne, Marion M., MD
Horton, Edward S., MD
Hoyer, Paul J., MD
Hudock, Jude, MD
Husson, Michael Andrew, MD
Innes, Susan A., MD
Jarett, Leonard, MD
Kamazon, Nelly, MD
Kellogg, Kim A., MD
Klenk, Philip J., MD
Konrad, Robert J., MD
Kowalyshyn, Michael J., MD
Kwa, Daniel M.S.K., MD
Lapeyre, Elizabeth A., MD
Lee, Jean H., MD
Levin, Norman P., MD
LiVolsi, Virginia A., MD
Lindenmayer, A. Earle, MD
Looby, Catherine R., MD
Maenza, Ronald M., MD
Manhoff, Clon T., MD
McCormick, John F., MD
McCue, Peter A., MD
McFarland, Miles M., MD
Meilhofer, Mary A., MD
Miettinen, Markku M., MD
Miller, Barbara A., MD
Millios, Alberto, MD
Mirchandani, Haresh G., MD
Mirchandani, Ila H., MD
Montone, Kathleen T., MD
Nadkarni, Rajani Arvind, MD
Naiken, Veerasamy S., MD
Naijar, Denise Dertane, MD
Nelson, Beverly P., MD
Newton, Sonia Y., MD
Olkowska, Danuta, MD
Peters, Sara B., MD, PhD
Peterson, Robert O., MD PhD
Powell, Alvin P., MD
Prestipino, Anthony Joseph, MD
Quinn, Dianne M., MD
Quirolgico, Benjamin P., MD
Raab, Carol R., MD
Rhim, Jonathan A., MD
Rorke, Lucy B., MD
Rose, David I., MD
Rubin, Emanuel, MD
Ruckdeschel, Richard H., MD
Rupp, Michael J., MD
Russo, Irma H., MD
Russo, Jose, MD
Sacheteva, Rajeev M., MD
Sainz, Irma M., MD
Salazar, Hernando, MD
Samlin, Barry, MD
Sano, Machteld E., MD
Sawhill, David L., MD
Schainker, Bruce A., MD
Schiffman, Raymond J., MD
Sekula-Pertman, Adrienne, MD
Sloan, Steven R., MD, PhD
Soldo, Catherine R., MD
Sunderman, F. William, MD
Svarstduk, Andrij E., MD
Urquhart, Marion Lena, MD
Uy-Putong, Antonia A., MD
Velez Rivera, Carlos A., MD
Vong, Garen T., MD
Wang, Tsaling, MD
Wartell, Michael J., MD
Warren, William J., MD
Weiss, Charles, MD
Wells, Gregg B., MD, PhD
Wolk, John Howard, MD
Woo, Zungpah, MD
Yaghsezian, Rouben, MD
Young, Nancy A., MD
Zangwill, Bruce C., MD
Zappi, Marcelo E., MD
Zarva, Loukia, MD
Zhang, Peishu, MD
Zimmerman, Michael R., MD

Philadelphia
Simpkins, Henry, MD

Phillipsburg
Saleh, Ahmed Nabil, MD

Phoenixville
Cook, Donald R., MD
Sugiura, Henry T., MD
Urban, Clifford H., MD

Pittsburgh
Abdulla, Mohamed A., MD
Aburano, Aldo, MD
Amortegui, Antonio J., MD
Anderegg, Katherine A., MD, PhD
Barnes, E. Leon, Jr., MD
Barach, Michael J., MD, PhD
Berman, Howard J., MD
Berman, Michael Anthony, MD
Block, Robert C., MD
Borochowitz, Dennis, MD
Bradley, Robert, MD
Brancazio, Lisa A., MD
Brandon, John M., MD
Breitfeld, Volker, MD
Calaas, Myrvas J., MD
Charnew, Irwin M., MD
Clarke, Martha R., MD
Cohen, Martin, MD
Compare, Ezio G., MD
Cooper, David Lynn, PhD, MD
Cooper, Jeanne A., MD
Dawson, Robert R., MD
DeLeon, Manuel R., MD
Dhir, Rajiv, MD
Dickman, Paul S., MD
Dunn, Jeanette C., MD
Edberg, Sanford H., MD
Edelstein, Joseph M., MD
Fernandes, Sheila P., MD
Fisher, Edwin R., MD
Fox, Karl R., MD
Geyer, Stanley J., MD
Glaiamas, Anthony J., MD
Hammad, Azzam, MD
Hartsock, Robert J., MD
Hillwig, Robert J., MD
Hoover, William W., MD
Hsu, Chia C., MD
Israel, Michael, MD
Ives, Nadine S., MD
Jasnosz, Katherine M., MD
Jones, William C., MD
Kapadia, Silloo B., MD
Kaplan, Sandra S., MD
Khasnabis, Sukamal, MD
Kilonsky, Bernard L., MD
Kotwal, Nirmal D., MD
Kurtz, John E., MD
Lavitt, Susanna S., MD
Locker, Joseph D., MD, PhD
Ludin, John W., MD
MacPherson, Trevor A., MD
Mahidhara, Seshu, MD
Manack, Leo, MD
Martinez, A. Julio, MD
Marwah, Shashi S., MD
McCarty, Kenneth S., Jr., MD
Mendlowski, Jerry J., MD
Miller, Thomas R., MD
Mitra, Bilma K., MD
Mostofizadeh, Mahpareh S., MD
Myers, Leonard B., MD
Nalesnik, Michael A., MD
Nalesnik, Oliver Kimka, MD
Nichols, Lawrence C., MD
Nine, Jeffrey Scott, MD
Norbut, Alan M., MD
Obort, N. Paul, MD
Peel, Robert L., MD
Pena, Carlos E., MD
Pizarro, Greg S., MD
Quintana, Paulina G., MD
Rabkin, Michael Scott, MD, PhD
Randhawa, Parmjeet Singh, MD
Rao, Gutti R., MD
Rao, Uma N.M., MD
Reed, W. Glenn, MD

Inspectors for LAP printed in bold type.

Pittsburgh (cont.)
Reddiford, Howard E., MD
Richert, Charles A., MD
Sahni, Ramona, MD
Saito, Reisuke, MD
Schwartz, Michael Alan, MD
Sethi, Pradeep K., MD
Singh, Gurmukh, MD, PhD
Sloman, Andrew J., MD
Smith, William L. Jr., MD
Song, Kyo Chul, MD
Stachura, Irene, MD
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WEATHERALL, D. J., Clinical Medicine (Magd.)

Faculty of English Language and Literature:

BAYLEY, J. O., English Literature (S. Cat.)
CAREY, J., English Literature (Mert.)
GRAY, D., English Language and Literature (LMH)
JONES, E. L., English Literature (New)
LEVI, P. C. T., Poetry (S. Cat.)
ROMAINE, S., English Language (Mert.)
STANLEY, E. G., Anglo-Saxon (Pemb.)

Fine Art

HOUSE, J. P. H., Slade Professor

Inter-faculty Committee for Japanese Studies:

STOCKWIN, J. A. A., Modern Japanese Studies (S. Ant.)

Faculty of Law:

ATIYAH, P. S., English Law (S. Joh.)
BROWNLIE, I., Public International Law (All S.)
DWORKIN, R. M., Jurisprudence (Univ.)
HONORÉ, A. M., Civil Law—Regius (All S.)
RAZ, J., Philosophy of Law (Ball.)
RUDDEN, B. A., Comparative Law (Bras.)
TWEEDDALE, G. H., English Law (All S.)

Faculty of Literae Humaniores:

ACKRILL, J. L., History of Philosophy (Bras.)
BOARDMAN, J., Classical Archaeology and Art (Linc.)
DAVIES, A. E., Comparative Philology (Som.)
DUMMETT, M. A. E., Logic (New)
FORREST, W. G. G., Ancient History (New)
HARRISON, R. M., Archaeology of the Roman Empire (Linc.)
LEWIS, D. M., Ancient History (Ch. Ch.)
LLOYD-JONES, P. H. J., Greek—Regius (Ch. Ch.)
MACINTYRE, A. J., Mathematical Logic (Mert.)
MILLAR, F. G. B., Ancient History (Bras.)
NISBET, R. G. M., Latin Language and Literature (Corp.)
PEASE, D. F., Philosophy (Ch. Ch.)
RUSSELL, D. A. F. M., Classical Literature (S. Joh.)
STRAWSON, Sir PETER, Metaphysical Philosophy (Magd.)

Faculty of Mathematics:

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BENJAMIN, T. B., Natural Philosophy (Qu.)
BIRCH, B. J., Arithmetic (Bras.)
DONALDSON, S. K., Mathematics (S. Ann.)
HOARE, C. A. R., Computation (Wolfs.)
JAMES, I. M., Geometry (New)
MORTON, K. W., Numerical Analysis (Ball.)
PENROSE, R., Mathematics (Wadh.)
QUILLEN, D. G., Pure Mathematics (Magd.)
WOODS, L. C., Mathematics (Theory of Plasma) (Ball.)

Faculty of Mediaval and Modern Languages:

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GRAYSON, C., Italian Studies (Magd.)
HARRIS, R., General Linguistics (Worc.)
MANGO, C., Byzantine and Modern Greek Language and Literature (Exeter)
MICHAEL, I. D. L., Spanish Studies (Exeter)
POSNER, R., Romance Languages (S. Hug.)

Faculty of Modern History:

HASKELL, F. J. H., History of Art (Trin.)
HOWARD, Sir MICHAEL, Modern History—Regius (Oriel)
LEYSER, K. J., Medieval History (All S.)
MATHIAS, P., Economic History (All S.)
MONTGOMERY, D., American History (Qu.)
O'NEILL, R. J., History of War (All S.)
PLATT, D. C. M., History of Latin America (S. Ant.)
POLE, J. R., American History and Institutions (S. Cat.)
ROBINSON, R. E., History of the British Commonwealth (Ball.)
STONE, N., Modern History (Worc.)

Inter-faculty Committee for Modern Middle Eastern Studies:

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Faculty of Music:

(vacant)

Faculty of Oriental Studies:

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BARR, J., Hebrew—Regius (Ch. Ch.)
DOWSETT, C. J. F., Armenian Studies (Pemb.)
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MADELUNG, W. F., Arabic (S. Joh.)
MATILAL, B. K., Eastern Religions and Ethics (All S.)
VAN DER LOON, P., Chinese (Univ.)

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ALLEN, K. W., Nuclear Structure (Ball.)
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BLACKWELL, D. E., Astronomy (New)
BRADY, J. M., Information Engineering (Keble)
CARRINGTON, A., Physical Chemistry (Royal Society Research) (Jesus)
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DALITZ, R. H., Research Professor of the Royal Society, Theoretical Physics (All S.)
DEWEY, J. F., Geology (Univ.)
ELLIOTT, R. J., Physics (New)
HIRSCH, Sir PETER, Metallurgy (S. Edm.)
McCONNELL, J. D. C., Physics and Chemistry of Minerals (S. Hug.)
MARCH, N. H., Theoretical Chemistry (Univ.)
MITCHELL, E. W. J., Experimental Philosophy (Wadh.)
PAIGE, E. G. S., Electrical Engineering (S. Joh.)
PERKINS, D. H., Elementary Particle Physics (S. Cat.)
ROWLINSON, J. S., Physical Chemistry (Exeter)
SANDARS, P. G. H., Experimental Physics (Ch. Ch.)
SCHULTZ, D. L., Mechanical Engineering (S. Hug.)
TURNER, D. W., Electron Spectroscopy (Ball.)
WILLIAMS, R. J. P., Royal Society Napier Research Professor, Inorganic Chemistry (Wadh.)
WROTH, C. P., Engineering Science (Bras.)

Faculty of Physiological Sciences:
- BLAKEMORE, C. B., Physiology (Magd.)
- BROWNLEE, G. G., Chemical Pathology (Linc.)
- GARDNER, R. L., Pathology, Royal Society Research (Ch. Ch.)
- GUILLERY, R. W., Anatomy (Hertf.)
- MATTHEWS, P. B. C., Sensorimotor Physiology (Ch. Ch.)
- NOBLE, D., Cardiovascular Physiology (Ball.)
- SMITH, A. D., Pharmacology (L. M. H.)

Faculty of Psychological Studies:
- BRYANT, P. E., Psychology (Wolfs.)
- COWEY, A., Physiological Psychology (Linc.)
- WEINKRANTZ, L., Psychology (Magd.)

Faculty of Social Studies:
- BRUS, W., Modern Russian and East European Studies (Wolfs.)
- COHEN, G. A., Social and Political Theory (All S.)
- HALSEY, A. H., Social and Administrative Studies (Nuff.)
- HAZLEWOOD, A. D., Commonwealth Studies, Research Professor (Pemb.)
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- MIRRLEES, J. A., Economics (Nuff.)
- NICKELL, S. J., Economics (Nuff.)
- PETERS, G. H., Agricultural Economics, Research Professor (Wolfs.)
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- SEN, A. K., Political Economy (All S.)
- SHAFER, B. E., American Government (Nuff.)

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Faculty of Theology:
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- O'DONOVAN, Rev. O. M. T., Moral and Pastoral Theology (Ch. Ch.)
- SANDERS, E. P., Exegesis of Holy Scripture (Qu.)
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- WILES, Rev. M. F., Divinity—Regius (Ch. Ch.)
- WILLIAMS, R. D., Divinity (Ch. Ch.)

READERS

Faculty of Anthropology and Geography:
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- NYE, P. H., Soil Science (S. Cross)
- PHILLIPSON, J., Animal Ecology (Linacre)

Faculty of Clinical Medicine:
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- TARIN, D., Pathology (Green)

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- MCINTYRE, D. I., Educational Studies (Jesus)

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- MCKENZIE, D. F., Textual Criticism (Pemb.)
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- STALLWORTHY, J. H., English Literature (Wolfs.)
- WEAVER, M. L. H. L., American Literature (Linacre)

Faculty of Law:
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- FINNIS, J. M., Laws of the British Commonwealth and United States (Univ.)
- HOOD, R., Criminology (All S.)
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- COULTON, J. J., Classical Archaeology (Mert.)
- MCGINN, C., Mental Philosophy (Corp.)
- WILKIE, A. J., Mental Philosophy (Wolfs.)

Faculty of Mathematics:
- EDWARDS, D. A., Mathematics (Linc.)
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- SEGAL, G. B., Mathematics (S. Cat.)

Faculty of Medieval and Modern Languages:
- RATTI, A. W., French Literature (Magd.)
- SEIFFERT, L., German (Hertf.)

Faculty of Modern History:
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- DICKSON, P. G. M., Modern History (S. Cat.)
- O'BRIEN, P. K., Economic History (S. Ant.)
- RAYCHAUDHURI, T., Modern South Asian History (S. Ant.)
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Faculty of Oriental Studies:
- VERMES, G., Jewish Studies (Wolfs.)
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- GRACE, M. A., Nuclear Physics (Ch. Ch.)
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- TAYLOR, F. W., Atmospheric Physics (Jesus)
- WHELAN, M. J., Physical Examination of Materials (Linacre)

Faculty of Physiological Sciences:
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- GORDON, S., Experimental Pathology (Exeter)
- PORTERFIELD, J. S., Bacteriology (Wadh.)
- POWELL, T. P. S., Neuroanatomy (S. Joh.)

Faculty of Psychological Studies:
- ARGYLE, J. M., Social Psychology (Wolfs.)

Faculty of Social Studies:
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- BLISS, C. J., International Economics (Nuff.)
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- HOOD, R. G., Criminology (All S.)
- JOHNSON, N., Comparative Study of Institutions (Nuff.)
- KASER, M. C., Economics (S. Ant.)
- ROBERTS, E. A., International Relations (S. Ant.)
- RYAN, A. J., Politics (New)
- WILSON, B. R., Sociology (All S.)

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WHITE, C. J. (Worc.)

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PERRINS, C. M. (Wolfs.)

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ROBERTS, A. G. (Wolfs.)

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ELLIS, T. M. R. (Ch. Ch.)

Forestry Institute:
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Queen Elizabeth House:
CARRIN, R. H. (S. Ant.)

Social and Administrative Studies:
HALSEY, A. H. (Nuff.)

Transport Studies Unit:
GOODWIN, P. B. (Linacre)

COLLEGES

All Souls College: Oxford, OX1 4AL; tel. (0865) 279379; f. 1438; for Fellows only; Warden P. M. FRASER (acting).

Balliol College: Oxford, OX1 3BJ; tel. (0865) 277777; f. 1263; Master A. J. P. KENNY.

Brasenose College: Oxford, OX1 4AJ; tel. (0865) 277830; f. 1509; Principal J. K. B. M. NICHOLAS.

Christ Church: Oxford, OX1 1DP; tel. (0865) 276150; f. 1546; Dean The Very Rev. R. W. HEATON.

Corpus Christi College: Oxford, OX1 4JF; tel. (0865) 276700; f. 1517; Pres. K. V. THOMAS.

Exeter College: Oxford, OX1 3DP; tel. (0865) 279600; f. 1314; Rector The Rt. Hon. Lord CROWTHER-HUNT.

Green College: Oxford, OX2 6HG; tel. (0865) 274770; f. 1979, for graduates; Warden Sir JOHN WALTON.

Hertford College: Oxford, OX1 3BW; tel. (0865) 279400; f. 1874; Principal Sir GEOFFREY WARNOCK.

Jesus College: Oxford, OX1 3DW; tel. (0865) 279707; f. 1571; Principal P. M. NORTH.

Keble College: Oxford, OX1 3PG; tel. (0865) 272727; f. 1870; Warden C. J. R. BALL.

Lady Margaret Hall: Oxford, OX2 6QA; tel. (0865) 274000; f. 1878; Principal D. M. STEWART.

Linacre College: Oxford, OX1 1SY; tel. (0865) 271650; f. 1962, as Linacre House, for graduates; Principal J. B. BAMBOROUGH.

Lincoln College: Oxford, OX1 3DR; tel. (0865) 279800; f. 1427; Rector The Rev. V. H. H. GREEN.

Magdalen College: Oxford, OX1 4AU; tel. (0865) 276000; f. 1458; Pres. K. B. GRIFFIN.

Merton College: Oxford, OX1 4JD; tel. (0865) 276310; f. 1264; Warden J. M. ROBERTS.

New College: Oxford, OX1 3BN; tel. (0865) 248451; f. 1379; Warden H. MCGREGOR.

Nuffield College: Oxford, OX1 1NF; tel. (0865) 278500; f. 1937, for graduates; Warden M. G. BROCK.

Oriel College: Oxford, OX1 4EW; tel. (0865) 276555; f. 1328; Provost The Rt Hon. Sir ZELMAN COWEN.

Pembroke College: Oxford, OX1 1DW; tel. (0865) 276444; f. 1624; Master Sir ROGER BANNISTER.

Queen's College, The: Oxford, OX1 4AW; tel. (0865) 279138; f. 1340; Provost The Rt Hon. Lord BLAKE.

St Anne's College: Oxford, OX2 6HS; tel. (0865) 274800; f. 1893 (as Society of Oxford Home Students); Principal Mrs C. D. T. PALLEY.

St Antony's College: Oxford, OX2 6JF; tel. (0865) 59651; f. 1950, for graduates; Warden A. R. M. CARR.

St Catherine's College: Oxford, OX1 3UJ; tel. (0865) 249641; f. 1868, reconstituted as a full College 1962; Master The Rt Hon. Sir PATRICK NAIRNE.

St Cross College: Oxford, OX1 3LZ; tel. (0865) 278490; f. 1965, for graduates; Master G. H. STAFFORD.

St Edmund Hall: Oxford, OX1 4AR; tel. (0865) 279000; f. c. 1278; Principal J. C. B. GOSLING.

St Hilda's College: Oxford, OX4 1DY; tel. (0865) 276884; f. 1893; Principal Mrs G. M. MOORE.

St Hugh's College: Oxford, OX2 6LE; tel. (0865) 274900; f. 1886; Principal Miss M. R. TRICKETT.

St John's College: Oxford, OX1 3JP; tel. (0865) 277300; f. 1555; Pres. Sir JOHN KENDREW.

St Peter's College: Oxford, OX1 2DL; tel. (0865) 278900; f. 1929 as St Peter's Hall; Master G. E. AYLMER.

Somerville College: Oxford, OX2 6HD; tel. (0865) 270600; f. 1879; Principal Miss D. M. S. D. PARK.

Trinity College: Oxford, OX1 3BH; tel. (0865) 279900; f. 1554; Pres. The Rt Hon. Lord QUINTON.

University College: Oxford, OX1 4BH; tel. (0865) 276602; f. 1249; Master K. BREWSTER.

Wadham College: Oxford, OX1 3PN; tel. (0865) 277900; f. 1612; Warden Sir CLAUS MOSER.

Wolfson College: Oxford, OX2 6UD; tel. (0865) 274100; f. 1965, for graduates; Pres. Sir RAYMOND HOFFENBERG.

Worcester College: Oxford, OX1 2HB; tel. (0865) 278300; f. 1714; Provost The Rt Hon. Lord BRIGGS.

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Campion Hall: Oxford, OX1 1QS; tel. (0865) 240861; f. 1896; Master Rev. P. HACKETT.

Greyfriars: Oxford, OX4 1SB; tel. (0865) 243694; f. 1910; Warden The Very Rev. T. M. MANN.

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Regent's Park College: Oxford, OX1 2LB; tel. (0865) 58287; f. 1810; Principal Rev. B. R. WHITE.

St Benet's Hall: Oxford, OX1 3LN; tel. (0865) 515006; f. 1897; Master Rev. P. D. HOLDSWORTH.

ATTACHED INSTITUTES

Maison Française: see under Learned Societies.

NERC Institute of Virology: see under Research Institutes.

Templeton College, Oxford Centre for Management Studies: see under Colleges.

Oxford Institute for Energy Studies: 29 New Inn Hall St, Oxford, OX1 2DX; f. 1983; Dir R. E. MABRO.

Centre for Postgraduate Hebrew Studies: 45 St Giles, Oxford, OX1 3LW; Pres. D. PATTERSON.

Centre for Socio-Legal Studies: Wolfson College, Oxford, OX2 6UD; Dir D. R. HARRIS.

Queen Elizabeth House: 21 St Giles, Oxford, OX1 3LA; f. 1954; development studies centre.

Ian Ramsey Centre: St Cross College, Oxford, OX1 3LZ; Dir A. R. PEACOCK.

Oxford Centre for Islamic Studies: see under Research Institutes.

UNIVERSITY OF READING

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Vice-Chancellor: E. S. PAGE

Deputy Vice-Chancellor: Prof. J. WRIGLEY

Pro-Vice-Chancellor: Prof. C. R. W. SPEDDING

Registrar: T. BOTTOMLEY

Librarian: J. THOMPSON

Library: see Libraries

Number of teachers: 650, including 84 professors

Number of students: 6,408

Publication: *Proceedings of the University* (annually).

DEANS

Faculty of Letters and Social Sciences: Prof. K. DAVIES

Faculty of Science: Prof. G. W. A. FOWLES

Faculty of Agriculture and Food: Prof. J. S. MARSH

Faculty of Urban and Regional Studies: Prof. A. W. EVANS

School of Education: Prof. R. WILSON (Chair.)

PROFESSORS

(some professors serve in more than one faculty)

Faculty of Letters and Social Sciences:

BIDDISS, M. D., History

CAMPBELL, P. W., Politics

CARRON, M. C., Economics

COUPE, W. A., German

DAVIES, J. C. H., Sociology

DAVIES, K., Law

DAVIES, M. C., French

DAVIS, R., Psychology

DOWNES, J. K., History of Art

DUNNING, J. H., International Investment and Business Studies

DUNSBY, J. M., Music

EMDEN, W. G. VAN, French

EVANS, A. W., Environmental Economics

FROY, M., Fine Art

GREGORY, R. G., Politics

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HUPTON, O., History

JACKSON, P., Law

KNOWLSON, J. R., French

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SALVESEN, C. G., English

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WARBURTON, D. M., Psychology

WILKINS, D. A., Applied Linguistics

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ADDIS, T. R., Computer Science

ALLEN, J. R. L., Geology

ALMOND, J. W., Microbiology

ATKINS, A. G., Mechanical Engineering

BAILEY, D. K., Geology

BAKER, K. D., Computer Science

BASSETT, D. C., Physics

BEECH-SMITH, D., Organic Chemistry

CURNOW, R. N., Applied Statistics

DICKINSON, H. G., Plant Cell Genetics

DILS, R. R., Physiology & Biochemistry

DUNN, P. D., Engineering Science

EMDEN H. F. VAN, Applied Entomology

EVANS, T., Physics

FELLGETT, P. B., Cybernetics

FOWLES, G. W. A., Inorganic Chemistry

FREY, H. M., Physical Chemistry

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HEYWOOD, V. H., Botany

HOSKINS, B. J., Meteorology

HUNT, J. N., Applied Mathematics

JENNINGS, B. R., Physics

LOWRY, P. J., Physiology and Biochemistry

MCCORMIC, C. W., Physics

MEAD, R., Applied Statistics

MILLS, I. M., Chemical Spectroscopy

MOORE, D. M., Botany

NASH-WILLIAMS, C. ST J. A., Pure Mathematics

NEWMAN, R. C., Physics

NURSTEN, H. E., Food Science

PEARCE, R. P., Meteorology

SEWELL, M. J., Applied Mathamatics

SIMKISS, K., Zoology

WILD, A., Soil Science

WRIGHT, J. D. M., Pure Mathematics

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GILES, A. K., Farm Management

HARVEY, D. R., Agricultural Economics and Management

MARSH, J. S., Agricultural Economics and Management

MORRIS, T. R., Agriculture

E X H I B I T S

marked at the

deposition of

EDWARD B. ILGREN

on

JUNE 22, 1994

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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

This Document Relates To:

UNITED STATES DISTRICT COURT
FIFTH DIVISION
DISTRICT OF MINNESOTA

CONWED CORPORATION,

Plaintiff,

vs.

Case No.: Civ. 5-91-88

UNION CARBIDE CHEMICALS AND PLASTICS COMPANY, INC. (f/k/a Union Carbide Corporation),

Defendant,

and

UNION CARBIDE CHEMICALS AND PLASTICS COMPANY, INC. (f/k/a Union Carbide Corporation),

vs.

OWENS-CORNING FIBERGLAS CORPORATION, WALKER JAMAR COMPANY, A.W. KUETTEL & SONS, INC., API, INC., AND MACARTHUR COMPANY,

Third-Party Defendants.

Ilgren
EXHIBIT NO. 11
6/22/94 L METZ

UNION CARBIDE CHEMICALS AND PLASTICS COMPANY, INC.'S PRELIMINARY STATEMENT REGARDING EXPERT WITNESSES

NOW COMES Union Carbide Chemicals and Plastics Company, Inc., and its attorneys, and for its preliminary disclosure

concerning expert witnesses, provides the following objections and information:

GENERAL OBJECTIONS

Union Carbide is not in a position at this point in the case to provide a definitive list of expert witnesses, nor to provide a complete list of the facts, opinions, or subject matter on which any experts it calls may testify. Plaintiff Conwed Corporation has still not furnished reports from two of its three experts, and Union Carbide still has not received a portion of Art Langer's report. Union Carbide has not had an opportunity to depose two of Conwed's experts, and has not finished the deposition of the third. Consequently, Union Carbide does not know exactly what testimony or opinions Conwed may offer, nor what work it experts may produce. Accordingly, Union Carbide cannot at this point know exactly what it will ask its experts to do in connection with this case, and so Union Carbide reserves the right to amend this disclosure by adding or deleting expert witnesses, or the scope of their testimony, as developments in the case warrant.

Without waiving these general objections, Union Carbide can provide the following information about its experts as of this time:

I. PULMONOLOGISTS

A. Mitchell G. Kaye, M.D.
Minnesota Lung Center
Suite 700
920 East 28th Street
Minneapolis, MN 55407

Dr. Kaye is a medical doctor and a pulmonologist. He

will testify, based upon his review of the medical records, x-rays, test results, and other material (possibly including an independent examination), and in light of his training and experience, concerning whether certain former employees at the Conwed plant suffer any asbestos-related injury. Dr. Kaye may also be asked to state the extent to which any employee who has an asbestos-related condition has been disabled under Minnesota's worker's compensation law, to make assessments of the extent of any such disability, and to comment on the limitations (if any) that such condition would place upon the former employee.

Dr. Kaye may also be asked to testify to general medical knowledge concerning the body and the lungs, and to general knowledge concerning how the body interacts with asbestos. Dr. Kaye may also be asked to give opinions concerning the prognosis for any particular Conwed worker, or for the group of workers.

B. Dr. William Weiss, M.D.
3912 Netherfield Road
Philadelphia, PA 19129

Dr. Weiss received his Bachelors and his M.D. degrees from the University of Pennsylvania. He was a Fellow of the College of Chest Physicians, and is a Fellow of the American College of Physicians. In addition to being a pulmonologist, Dr. Weiss has trained as an epidemiologist, and has taught epidemiology. He is a Professor of Medicine, Emeritus of Hahnemann University Medical School in Philadelphia.

Based upon his training and experience, the scientific literature, information provided to him about the Conwed plant and

Conwed employees (or former employees), and information about Calidria, Dr. Weiss may be asked to give opinions concerning the extent of asbestos-related disease found in former Conwed workers; the role, if any, which Calidria asbestos played in producing that disease; the role which the amosite used in the plant played in producing that disease; the expected future incidence of asbestos-related disease in the group of former Conwed workers; and whether the disease that has been identified to this point is actually an asbestos-related disease or is caused by some other factor.

Dr. Weiss may also testify about general medical knowledge and general knowledge concerning asbestos and its interaction with the human body.

C. Dr. Hilton C. Lewinsohn, M.D., B.C.h., F.F.O.M., F.A.C.O.E.M., F.C.C.P. and D.I.H.
Center for Occupational and Environmental Health at Exeter Hospital, Inc.
108 High Street
Exeter, NH 03833

Based upon his training and experience, his review of the scientific literature, his evaluation of materials relating to the Conwed plant and to Calidria asbestos, Dr. Lewinsohn may be asked to give opinions upon the ability of Calidria asbestos to cause disease in workers under the conditions during which it was used at the Conwed plant, the role of amosite and the production of disease in the workers at Conwed, the extent to which former Conwed employees at Cloquet suffer from asbestos-related disease, the scientific literature regarding the ability of chrysotile and short-fibered chrysotile to cause disease, the appropriateness of the steps taken by Conwed's management from an occupational health

standpoint in light of the knowledge available to them, and the extent to which alleged health problems in former Conwed workers are attributable to factors other than asbestos. Dr. Lewinsohn is also a former employee of Union Carbide.

XI. PATHOLOGISTS

A. Dr. Ed B. Ilgren, M.A., M.D., D.P.H.I.L., M.A.C.P.A.T.H., M.R.C.P.A.T.H.
Conwyn Apartments, No. 503
830 Montgomery Avenue
Bryn Mar, PA 19010

Dr. Ilgren's testimony will be based upon his training and experience, his review of the scientific literature, his review of the opinions and testimony provided by plaintiff's experts, his review of materials relating to the Conwed plant, and his review of materials concerning Calidria asbestos.

Dr. Ilgren will testify on the biological effects of exposure to Calidria asbestos, and offer the opinion that of all types of chrysotile, Calidria is the least active. Moreover, he will testify that chrysotile uncontaminated with amphibole cannot induce mesothelioma. In the Conwed plant, lung cancer would either have been related to amosite exposure, smoking, or some other cause aside from Calidria asbestos. The putative mesotheliomas noted at Conwed would have been due to amosite exposure or a nonasbestos-related cause. Dr. Ilgren will show that pleural thickening is not specific for asbestos exposure, while pleural plaques, though possibly specific for asbestos, do not carry any future cancer risk nor impose any significant functional impairment. Dr. Ilgren will also discuss the biological difference between long and short fiber

using data from animal experiments, human epidemiological studies, and his review of the medical literature, especially as it relates to the mineralogical and physical properties of Calidria that distinguish it from other types of asbestos. Dr. Ilgren may also refute claims of plaintiff's experts on issues relating to these topics, and may also be asked to give diagnostic opinions concerning the medical status of individual cases of former Conwed employees, based upon the clinical and pathological materials available for review. In the course of this testimony, Dr. Ilgren may provide general medical and biological information upon which his opinions are based.

B. Allen Gibbs
Pathology Department
Landough Hospital
Penarth, Glamorgan
UK CFC 1 XX

If Dr. Gibbs is called, it will be to testify about the biological effects of exposure to Calidria asbestos, and compare and contrast that to the effects of exposure to other types of asbestos. Dr. Gibbs may be asked to give testimony based upon his personal experience concerning asbestos-related diseases in the United Kingdom. He may comment upon the biological differences between different types of fibers, based upon data reported in the medical literature, and upon materials gathered from his own work and observations.

Dr. Gibbs may also be asked to comment upon the opinions and work of plaintiff's experts.

III. INDUSTRIAL HYGIENISTS

A. Richard J. Lee, Ph.D.
R.J. Lee Group, Inc.
350 Hochberg Road
Monroeville, PA 15146

Dr. Lee obtained his Ph.D. in theoretical solid state physics. His specialty is analysis of materials with electron microscopy, quantitative electron diffraction techniques, automated electron diffraction pattern analysis techniques and automated techniques for combined x-ray microanalysis and electron microscopy.

If asked, Dr. Lee will review the work of plaintiff's experts as it relates to the composition of Calidria asbestos in materials taken from the KCAC mine. Dr. Lee may also be asked, as an industrial hygienist, to comment on any calculations of dust concentrations or asbestos concentrations in the atmosphere at the Conwed plant. He may also be asked to examine materials taken from the Conwed plant for their asbestos content.

B. Dr. Harrison Rhodes
c/o Kelley, Drye & Warren
101 Park Avenue
New York, NY 10178

Dr. Rhodes was employed by Union Carbide as an industrial hygienist, and in that position supervised Union Carbide's dust count program for Calidria customers until June 30, 1985.

If asked, Dr. Rhodes will give his opinion, based upon his experience, training, and information provided to him about the Conwed plant, concerning dust concentrations in the plant. Dr. Rhodes may also be asked to comment on any calculations of dust

concentrations made by plaintiff's experts. He is also knowledgeable concerning the dust counts which were taken at Union Carbide's mine and mill in California.

C. Dr. Robert Murray
c/o Kelley, Drye & Warren
101 Park Avenue
New York, NY 10178

Dr. Murray is a professor of industrial hygiene in England. He has extensive experience inspecting industrial facilities in the United Kingdom. He is familiar with, and can testify about, the historical development of industrial hygiene and safety practices employed by plant managers to protect their workers from asbestos when asbestos was used as an ingredient in the manufacturing process. If asked to testify, Professor Murray will explain that Conwed failed to take steps to protect the health of its workers from asbestos, despite the fact that such steps and techniques had been known and reported since at least the 1930's. Dr. Murray will also base his testimony upon the information regarding Conwed's knowledge and practices concerning asbestos.

D. Doug Neal
c/o Kelley, Drye & Warren
101 Park Avenue
New York, NY 10178

Mr. Neal is a retired industrial hygienist who previously worked for Union Carbide. He has extensive experience with asbestos, and is familiar with and can testify about the history of the use of asbestos in industrial facilities, and the history and development of industrial hygiene and safety practices related to that use. If asked to testify, Mr. Neal will express the opinion

that under good industrial hygiene practice, Conwed had the primary responsibility for insuring a safe place for its employees to work, and that Conwed failed to take basic steps to protect its workers.

IV. MINERALOGISTS

A. Mark Van Buelen
Harvard University
Cambridge, MA 02138

Mr. Van Buelen is a mineralogist who has extensively worked in and studied with the New Idria area in California. If asked to testify, Mr. Van Buelen will explain the geologic forces that produced the New Idria area, the Calidria asbestos deposit, the geological and mineralogical facts that make Calidria unique, and how those factors affect the final characteristics of Calidria asbestos. Mr. Van Buelen is also knowledgeable concerning other minerals found in the New Idria deposits, and will testify that there is no fibrous amphibole contamination in Calidria asbestos. Mr. Van Buelen also has samples of material taken from the King City mine, and may be asked to testify about that material and/or to critique the work of plaintiff's expert Art Langer.

B. Professor Fred Pooley
School of Engineering
Department of Mining & Minerals
University of Wales
P.O. Box 917
Cardiff, Wales CF21XH

Dr. Pooley is a mineralogist who has spent much of his career investigating the biological effects of various minerals, particularly asbestos. He is knowledgeable concerning the difference in biological impact between Calidria and other types of asbestos (including the difference between Calidria and other types

of chrysotile). Dr. Pooley may also testify about the differences in biological effects between short and long fibered asbestos fibers, and offer opinions on the lack of effect that Calidria would have had on the health of Conwed workers, and the fact that the amosite used in the plant is likely responsible for any asbestos-induced mesothelioma found in the working population. Dr. Pooley may also be asked to critique the work and opinions of plaintiff's experts.

C. Dr. Fred Mumpton
31 Sherwood Drive
Brockport, NY 14420

Plaintiff has already deposed Dr. Mumpton, and is familiar with his areas of expertise and his opinions. If called to testify, Dr. Mumpton will describe the nature of Calidria asbestos from a geological and mineralogical perspective, the nature of the New Idria deposit from which it is mined, and what makes Calidria asbestos unique. Dr. Mumpton may also be asked to testify concerning his work for Union Carbide.

Dated this 4th day of February, 1994.

FOLEY & LARDNER,
Attorneys for Union Carbide
Chemicals and Plastics Co., Inc.

Trevor J. Will

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Ilgren 12

A COMPARATIVE STUDY OF PULMONARY TUMORS FROM THE SAN DIEGO ZOOLOGICAL GARDENS AND THE TUMOR REFERENCE COLLECTION, IMPERIAL CANCER RESEARCH FUND, LONDON

E. B. ILGREN, L. GRINER
K. BENIRSCHKE, AND L. S. C. PANG

Spontaneous primary neoplasms of the lung are uncommon in many animal species.[1-5] The predominant human pulmonary tumors, the squamous-cell and oat-cell carcinomas,[6-9] are believed to be causally related to cigarette smoking, are more frequently seen in urban than rural areas, often metastasize early and widely, and represent a significant proportion of all types of human tumors. Similar histologic lesions are almost unknown to occur spontaneously in domestic and captive wild animals.

This paper examines in detail most of the 31 pulmonary tumors found in the captive wild animal collection of the San Diego Zoo (SDZ), as well as the 7 lung tumors from the Tumour Reference Collection (TRC) of the Imperial Cancer Research Fund, London. The TRC cases were received from comparative pathologists throughout the world, and have not been described previously.

The object of this study was to determine if tumors from either the TRC or SDZ series histologically resembled certain human tumors associated with exposure to different pulmonary carcinogens, such as cigarette smoke.

DESCRIPTION OF CASES

Reptiles

To date, few primary epithelial or mesenchymal pulmonary tumors have been found in reptiles.[10-13] There were 28 reptilian tumors in the SDZ series—12 percent of the total—but only 2 of these were found in the lung. The first case (SDZ-2595) was that of

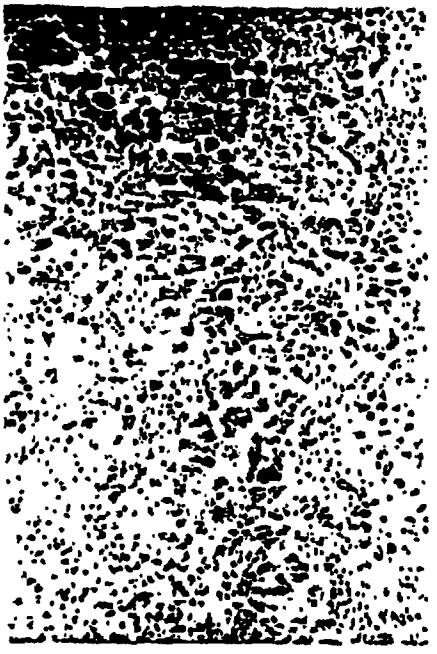

FIG. 1. Papillary adenocarcinoma extensively invading lung of Cape cobra (*Naja nivea*) H&E. x 213. (SDZ-2595).

a Cape cobra (*Naja nivea*) with a pulmonary adenocarcinoma that extensively invaded the lung parenchyma as well as the mediastinum. The tumor exhibited the cytologic features of a peripheral, bronchiolo-alveolar tumor (Fig. 1). The second case (SDZ-4538; AFIP 1343278) was that of a timber rattlesnake (*Crotalus horridus*) with a metastatic lymphosarcoma and an associated urate nephropathy.[14]

Birds

The mortality due to neoplasia in some species of fowl may approach 60 percent; most tumors found in birds are hematopoietic in origin.[1,11,16] In addition, Stewart and Ratcliffe found 19 primary pulmonary epithelial tumors in the Philadelphia study,[1,17] and at least 6 of these were shown to be malignant. As Campbell states, the primary pulmonary epithelial tumors of birds can be extremely difficult to distinguish from the bronchial epithelial hyperplasias associated with chronic inflammatory conditions.[15] The infrequency of avian pulmonary tumors is probably related to several factors.[18-22] For example, birds have a relatively long trachea, variable numbers of mucous glands, large air sacs, few lymphatics, a diffuse reticuloendothelial system, and a rapid respiratory rate. Such physiologic variables significantly influence the degree, type, and pattern of particle deposition within the avian lung. These differences could in turn account for the low frequency of avian pulmonary tumors which are histologically similar to those human neoplasias associated with the deposition of carcinogenic particulate matter.

In the present study, three avian tumors were examined. The first case, involving a wood partridge (*Rollulus rouloul* SDZ-10286), was found to be a small tumor arising from a major bronchus. There was no other evidence of endobronchial growth, nor any clear association with the overlying submucosal glands. Histologically, the tumor resembled the rare oncocytoid variant of a human carcinoid.[9] Although the argentaffin cell system from which the human carcinoids are thought to arise has also been demonstrated in mammals, birds, and some invertebrates, pulmonary carcinoids are rarely found in any nonhuman species.[2,24-26] The other two cases included that of a peaceful dove (*Geopelia placida* SDZ-8562), which died from a metastatic pancreatic, papillary adenocarcinoma histologically similar to its human counterpart,[27] and that of a blackwinged stilt (*Himantopus leucolophalus* SDZ-8958), which died from a metastatic squamous-cell carcinoma. The latter arose from a primary tumor associated with a chronic ulcer of the right wing. The origin of human cancers from sites of chronic inflammation, i.e., old burns and draining sinuses, is also well recognized.[28]

Mammals

Marsupials. Previously, only two other pulmonary tumors have been found in marsupials. These occurred in American opossums (*Didelphis marsupialis*) (PhZ-9980[2,29,30]), one of which died from a bronchogenic, epidermoid carcinoma, while the other (Lndn-64871[31]) expired with a bronchial adenoma. The single pulmonary tumor noted in this study arose from a major bronchus within the lung of another American opossum (SDZ-6864). The tumor itself was well circumscribed and glandular, with few mitoses. There was extensive inflammation and necrosis in the adjacent pulmonary parenchyma, but little evidence of invasion by tumor. Microscopically (Fig. 2), the neoplasm resembled one of the atypical pulmonary epithelial proliferations of humans.[9]

Edentata. The pulmonary lesion discovered in a nine-banded armadillo (*Dasypus novemcinctus* SDZ-10608) has been described in detail elsewhere.[32] The tumor was a benign, peripheral nodule that histologically resembled human focal adenomatosis.[9,33] The alveolar lining cells were replaced by mucus-secreting columnar epithelium, and there was no evidence of endobronchial infiltration.[33-36] Similar lesions have been artificially induced in the horse and rat with *Crotalaria retusa*,[35] in the rabbit with dibenzanthracene and rubidium-173,[36] and in the sheep by various means.[37-41]

Rodentia and Lagomorpha. With the exception of mice, spontaneous pulmonary tumors are relatively rare in rodents.[42-46] This is especially true of hamsters,[3,47-50] guinea pigs,[2,3,51] and rats.[43] Furthermore, guinea pigs and rabbits are particularly resistant to the development of induced lung tumors.[52] However, the hamster has, in contrast to its low frequency of spontaneous lung neoplasms, a relatively high incidence of induced squamous-cell tumors of the trachea and major bronchi.[53,54] Spontaneous lung tumors of rodents are usually peripheral, solitary adenomata,[2,3,55,56], and are much less common than secondary tumors such as metastatic lymphosarcomas. Furthermore, induced squamous-cell tumors are more common in the hamster and rat

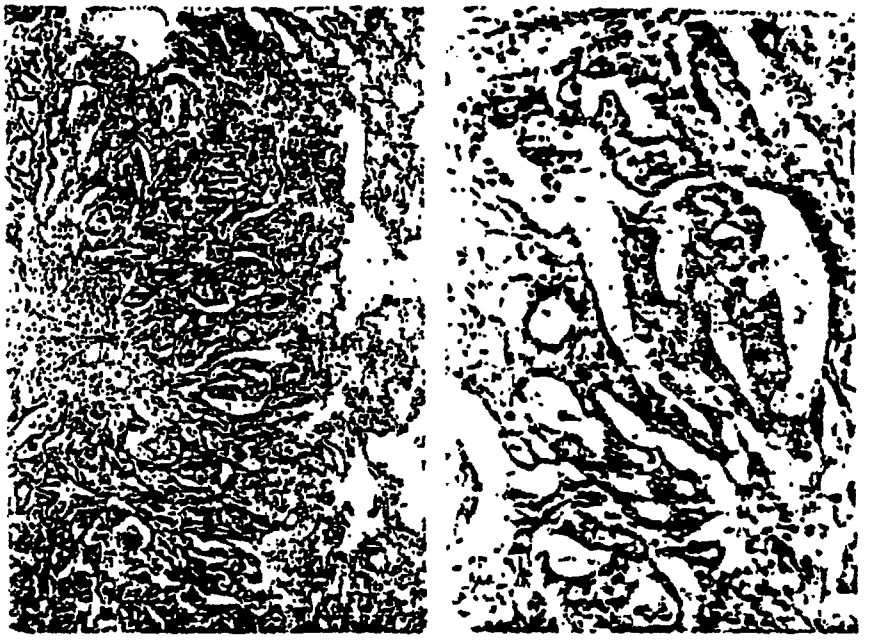

FIG. 2. Left. A pulmonary adenoma in an American opossum (*Didelphis marsupialis*) resembling an atypical epithelial proliferation of the human lung. H&E. ×55. Right. Detail of tumor to the right (SDZ-6864). H&E. ×213.

than in most rodents.[1,33,37-40] Although such squamous-cell neoplasms metastasize infrequently, metastasis, when it does occur, is usually to atypical sites such as the kidney, rather than to the liver and the lung, as in humans. The mouse is the only rodent with a relatively high incidence of spontaneous pulmonary tumors.[33] Such neoplasms are mostly subpleural and adenomatous. As in the rat, these tumors are rarely invasive, and less than 4 percent metastasize.[33,61] Only one adenocarcinoma was found in a mouse in the present study (TRC-1214). It was widely metastatic and invasive (Fig. 3). No oat-cell carcinomas and very few spontaneous squamous-cell carcinomas have been found in mice.[33,62,63] Thirty percent of the metastases from these rare, spontaneous murine carcinomas become sarcomatous.[61] Induced murine pulmonary tumors, in contrast to the spontaneous types, are usually multifocal, metastatic, and centrally-situated squamous-cell carcinomas.[33] Extensive genetic studies on murine pulmonary neoplasms have established a relationship between the susceptibility to developing pulmonary tumors and genes associated with certain morphologic characteristics, e.g., flexed, waved, and vestigial tails.[64] Pulmonary tumors are almost unknown in captive wild rodents other than the wild house mouse. No cases have been found in the Philadelphia or London Zoological collections. In this study, multiple, peripheral adenomata were found in an Antelope ground squirrel (*Citellus leucurus* SDZ-9413). These pulmonary lesions (SDZ-9413) (Fig. 4) histologically

FIG. 3. Papillary pulmonary adenocarcinoma in a laboratory mouse (TRC-1214). H&E. ×55. Note the extensive vascular and parenchymal invasion.

FIG. 4. Peripheral adenomata in the lung of an antelope ground squirrel (*Citellus leucurus*) Detail of tumor (SDZ-9413). H&E. ×213.

FIG. 5. Papillary adenoma in the lung of a laboratory mouse (*Mus musculus*) (TRC-6005). H&E. ×136.

resembled those seen in a laboratory mouse (TRC-6005) (Fig. 5) and a hamster (TRC-6008).

Carnivora. Neoplasms are found in 5 percent of all breeds of dog,[1,63-69] and the brachycephalic strains, such as the boxer, have the highest incidence.[53] Overall, however, pulmonary tumors are relatively rare in dogs, comprising from 1 to 3 percent of all canine neoplasms. The average age of dogs with pulmonary tumors is 10 years,[1,65] and there is no urban-rural variation in tumor incidence.[70] In one study of 16 lung tumors from over 10,000 dogs,[67] 12 tumors were bronchiolo-alveolar, and these failed to metastasize. The other 4 tumors in this series[67] were also glandular, but were associated either with foci of squamous metaplasia, or, more rarely, with squamous-cell carcinoma. Many induced lung tumors in dogs are alveolar, and these rarely metastasize beyond the hilar lymph nodes.[65,70] In the present study (TRC-2368), a 9-year-old male English setter died from a bronchogenic adenocarcinoma which had metastasized to the hilar and periaortic lymph nodes (Fig. 6). This animal's tumor appeared to arise from the bronchial epithelium, and was histologically similar to the feline tumor described below (cf. TRC-3334) (Fig. 7).[3]

Spontaneous lung tumors in cats are even less common than in dogs. They are usually subpleural, multifocal, and glandular.[71] Such tumors metastasize more frequently than those found in dogs, and squamous-cell carcinomas are practically unknown in this species. In contrast to these peripherally situated tumors, those found

FIG. 6. Papillary bronchogenic adenocarcinoma in an English setter dog (TRC-2368). H&E. x 272.

FIG. 7. Papillary bronchogenic adenocarcinoma invading pulmonary parenchyma in a domestic cat (TRC-3334). H&E. x 136.

FIG. 8. Bronchogenic adenocarcinoma in a domestic cat. Tumor appeared to arise from subepithelial glands (TRC-4308). H&E. × 136.

in cases TRC-3334 (Fig. 7), TRC-3266 and TRC-4388 (Fig. 8) arose from major bronchi, and all three were found in domestic cats. The first two were papillary adenocarcinomas, while the other was a secretory adenocarcinoma which arose from subepithelial glands. Although no squamous-cell carcinomas were seen in domestic cats, bronchiolar squamous metaplasia, a potentially premalignant lesion, was seen in one animal (TRC-3640).

Spontaneous primary pulmonary epithelial tumors are extremely rare in captive wild carnivores, and only one such case was found in the present study. This animal, a snow leopard (*Panthera uncia* SDZ-4795), died with multiple pulmonary adenomata. These unusual lesions were histologically similar to those just described in the domestic cat (TRC-3266), and were also of subepithelial glandular origin (Fig. 9). Several spontaneous secondary neoplasms were also found in the SDZ series. A maned wolf (*Chrysocyon brachyurus* SDZ-11576) died of a metastatic, papillary renal carcinoma not unlike that described in an earlier study. (See PhZ-19060[2].) Microscopically, these tumors resembled their human morphologic counterparts.[14,27] Another maned wolf (SDZ-10282) died from a metastatic fibrosarcoma arising from its right femur. Sarcomas of bones are particularly common in certain strains of domestic dogs (e.g., the Great Dane), and some have suggested that there is an association between extreme leg length, constant mechanical stress, and the development of hind-limb tumors. Although the maned wolf physically resembles these large domesticated strains of dogs, few tumors analogous to the ones found in the Great Dane have been

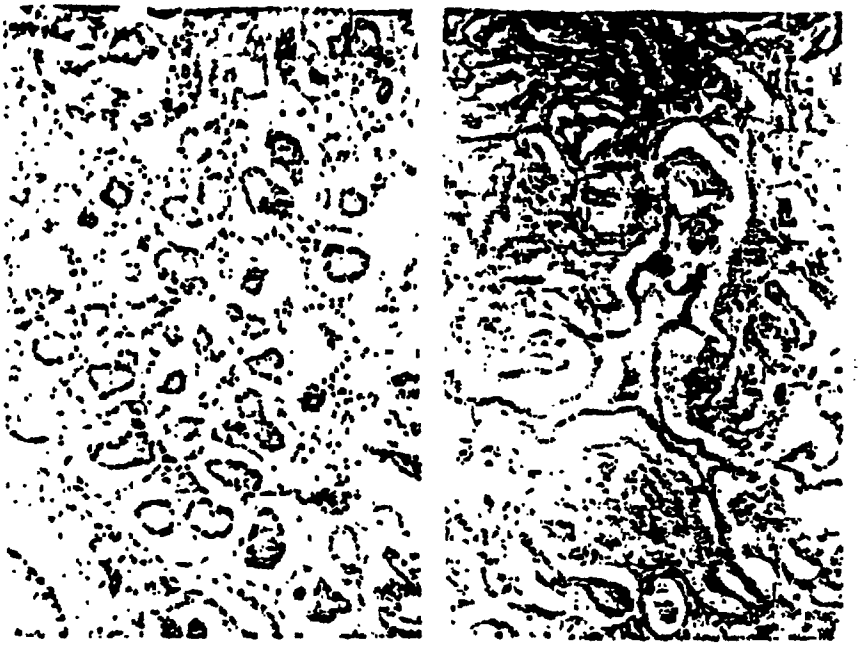

FIG. 9. Pulmonary adenomata arising from subepithelial glands in a snow leopard (*Panthera uncia*). Left shows normal subepithelial glands H&E. ×213. Right demonstrates dysplastic subepithelial glands (SDZ–4795). H&E. ×213.

reported in captive wild canines. The other tumors of wild dogs found in this study included a metastatic nasopharyngeal carcinoma (epithelioma) in a golden jackal (*Canis aureus* SDZ–10129); this tumor was morphologically similar to that seen in the human and in a Cape hunting dog (*Lycaon pictus* SDZ–11204) with a metastatic hemangiosarcoma. The latter probably arose from the aorta and was associated with primary carcinomas of the mamma, pancreas, and thyroid. Although aortic sarcomas in dogs are thought to be associated with *Spirocerca lupi*, no parasites were identified in this case.[72]

Artiodactyla. Although very few primary pulmonary tumors have been found in this order of mammals,[73–75] previous studies have been based largely on the examination of slaughtered animals killed before 8 years of age. Since most lung tumors in cattle have been found in animals older than 6 years of age, more tumors would obviously have been seen if these animals had been allowed to live out their natural life span, which is usually between 15 and 20 years.[76,77]

Few cases of primary squamous-cell carcinoma of the lung have been found in cattle.[78–80] The more common type of bovine lung tumor is a multifocal, scirrhous, peripheral adenocarcinoma with an alveolar infundibular pattern distinct from that

found in human pulmonary tumors.[81-83] This tumor metastasizes more often than do histologically similar lesions of the dog.[73] Most metastases, however, are confined to the regional lymph nodes, and extranodal spread is rare.[83] As in other mammals, secondary lung tumors in cattle are far more common than primary neoplasms. These usually arise either from carcinomas of the eye and the uterus, or from lymphosarcomas.[71,77]

Excluding Jaagsiekte, primary lung tumors are rare in domestic sheep[81,82] and in captive wild artiodactyls. In the present study, an impala (*Aepyceros melampus* SDZ-7823) was found with multiple scirrhous adenomata histologically similar to those found in domestic cattle (Fig. 10). A pulmonary adenocarcinoma was also seen in a springbok (*Antidorcas marsupialis* SDZ-10086). This animal's tumor (SDZ-10086) was not scirrhous, but was similar to the type of severe secondary bronchiolo-alveolar hyperplasia often associated with chronic pulmonary inflammation and consolidation. The lesion was, however, histologically distinct from the cellular variety of Jaagsiekte[84-86] and did not metastasize. This springbok (SDZ-10086) also exhibited marked joint swelling similar to that seen in birds,[13] dogs,[66,87,88] monkeys,[89] and humans with pulmonary hypertrophic osteoarthropathy.

A malignant spindle-cell and epithelioid mesothelioma was found in an eland (*Taurotragus oryx* SDZ-10125). Only two other mesotheliomas in captive wild mammals have been reported, one in a Cape hunting dog (Ph Z-9604) and the other in a clouded leopard (Ph Z-2516). However, the tumors in both of these cases were

FIG. 10. Scirrhous bronchiolo-alveolar adenoma in the lung of a Kenyan impala (*Aepyceros melampus*) (SDZ-7823). H&E. ×213.

localized, fibrous, and partially composed of osteoid, in contrast to the highly invasive neoplasm seen in this series (SDZ-10125). Such localized lesions may be similar either to the rare benign, fibrous mesotheliomas of humans or to certain reactive mesothelial proliferations often found adjacent to foci of parenchymal inflammation.[9] Asbestos particles were not seen in any of the cases (Ph Z or SDZ) described above.[30]

Tumors of the lung are very rare in domestic goats.[3] A wild goat (*Capra aegagrus* SDZ-6474) was found with multiple, peripheral, papillary bronchiolo-alveolar and bronchial hyperplastic nodules. Although some of these nodular lesions were associated with lung worms, this type of reactive bronchial epithelial change is usually not due to such parasites. The most common variety of bovine lung worm, *Dictyocaulus viviparus*, usually elicits an alveolar granulomatous inflammatory reaction with epithelialization of the alveolar lining cells.[31] Less commonly, these parasites may produce a secondary bronchiolitis that leads to consolidation and emphysema.

Two captive wild artiodactyls in this study had interesting secondary tumors. The first, an impala (*Aepyceros melampus* SDZ-8313), died from a metastatic adenocarcinoma that appeared to arise from the uterine cervix. Histologically, this tumor very closely resembled the rare human cervical adenocarcinoma of Wolffian or mesonephric duct origin—the well differentiated, tubular mesonephroma (Fig. 11).[32,33] Although endometrial carcinomas are well recognized in cattle,[1] mesonephroid tumors have not been described before in either domestic or captive wild species. The other secondary tumor in this series—a metastatic squamous-cell

FIG. 11. Pulmonary metastasis from cervical tumor of presumptive mesonephric duct origin in a Kenyan impala (*Aepyceros melampus*) (SDZ-8313). H&E. ×213.

carcinoma in a Wyoming pronghorn (*Antilocapra americana* SDZ-8957, AFIP 1502033)—arose from the skin of the lower eyelid, not unlike the so-called "cancer eye" of Hereford cattle.[73]

Pinnipedia. Little information exists concerning neoplasia in this order of mammals. No tumors were found in either the Philadelphia or London reports, although one tumor and one hamartoma were seen in this study. The first of these involved a California sea lion (*Zalophus californianus* SDZ-10092: AFIP 1548068) which died from a metastatic renal carcinoma. The tumor was a clear-cell variant microscopically similar to its human counterpart, which showed extensive venous invasion (Fig. 12). In the other case a Baikal seal (*Pusa sibirica* SDZ-3916) died with an associated ectopic pulmonary thyroid (Fig. 13). Although intrapericardial thyroid rests are well recognized in dogs,[36] deposits of ectopic thyroid near the lung are most unusual in any species including humans.

Proboscidea. Previously, only two pulmonary tumors, both of questionable malignancy, have been described in this order of mammals.[39,56] The case from the present study (SDZ-8085: AFIP 1472532) was that of an Asian elephant (*Elephas maximus*) that died with multiple pulmonary lymphoid nodules and no evidence of systemic disease. The lesions were composed mainly of mature lymphocytes and histiocytes arranged as germinal centers (Fig. 14 Left). There was little evidence of parenchymal invasion. This tumor, as well as the other two described elsewhere (v.s.), contained herpetic intranuclear inclusions within the bronchial epithelium (Fig. 14 Right). It is unlikely that this was a primary herpetic pneumonitis, for, at least in

FIG. 12. Pulmonary metastases from renal-cell carcinoma within the vessels and air spaces of the lung of a sea lion (*Zalophus californianus*) with a detail of the metastatic tumor showing "clear-cell" features (SDZ-10092). H&E. x 213.

FIG. 1:
adhere
seal (*Pu*

FIG. 1
maxim
Herpet

FIG. 13. Ectopic island of thyroid tissue adherent to anterior pleural surface in a Baikal seal (*Pusa sibirica*) (SDZ-3916). H&E. ×55.

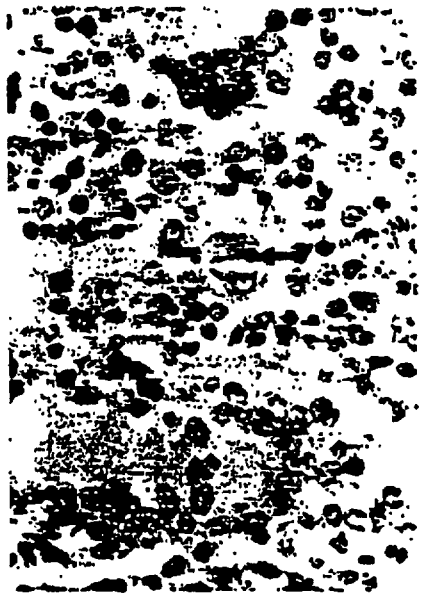

FIG. 14. Left. Pseudolymphoma (? lymphoid hyperplasia) in the lung of an Asian elephant (*Elephas maximus*) note germinal centers with lymphocytic parenchymal invasion. Giemsa. ×55. Right. Herpetic intranuclear inclusions within epithelial cells (one arrowed) (SDZ-8085). H&E. ×553.

humans, there would have been a more hemorrhagic, parenchymal necrosis. Human pulmonary pseudolymphomas, such as those described by Castleman, bear some resemblance to these lesions,[97,98] but are generally more diffuse. The differential diagnosis of pulmonary pseudolymphoma, lymphosarcoma, or severe, chronic, interstitial pneumonitis may be quite difficult. It can often only be made in retrospect, and the true diagnosis may remain uncertain unless resolution, recurrence after removal, or dissemination of disease occurs. Similar herpetic lymphoproliferative dysplasias and neoplasias are well recognized in birds, mammals, and humans,[99] although these disorders are rarely confined to one organ such as the lung.

Primates. Ratcliffe states that primates had the lowest tumor incidence of all mammals at the Philadelphia Zoo and, in over 32 years, he did not find one pulmonary neoplasm in this order of mammals.[17] No cases were found in the London series, although several lung tumors are described in other isolated studies.[100-102] The one case found in the SDZ series was that of a 22-year-old Guenon monkey (*Cercopithecus diana* SDZ-3473) which died with multiple peripheral pulmonary adenomata. There was neither parenchymal invasion nor metastases. The lesions histologically resembled the atypical epithelial proliferations of the human lung described by Liebow.[9]

DISCUSSION AND CONCLUSIONS

The present report supports the rarity of pulmonary tumors in animals. There is, moreover, a total absence in this study of any tumor histologically similar to the smoking-related human lung cancers, namely, the primary pulmonary squamous-cell, anaplastic, and oat-cell carcinomas. The animal lung tumors described herein may be histologically, and even biologically, similar to their human counterparts—the bronchiolo-alveolar carcinomas, adenomas, adenomatoses, bronchial glandular tumors, and carcinoids. In general, the most common types of animal lung tumors are adenomas and, less frequently, adenocarcinomas. These tumors are usually multifocal, peripheral, infiltrative,[38] and of two major types. The first, a papillary variety, is the most common, and is especially frequent in domestic animals. It may be scirrhous and contain foci of osteochondral metaplasia. The other is a bronchiolo-alveolar variant and may also be slightly papillary. Only 4 percent of tumors of the second type metastasize.[43] Although these less common alveolar tumors have been extensively studied in both humans and laboratory animals, confusion still exists as to their possible infectious nature, as well as to their precise cell of origin.[77,103-113] In veterinary practice, animals are either killed prematurely or cannot be observed continuously. The frequent diagnostic clinical information so helpful in human medicine is therefore often not available to the comparative pathologist. For this reason, it is frequently impossible to precisely diagnose a great number of animal tumors, since many of these neoplasias cannot be properly evaluated by histologic criteria alone. Furthermore, studies based on abattoir material may miss primary tumors, e.g., adenocarcinomas of the uterus[114] or squamous-cell carcinomas of the eyelid, which not infrequently metastasize to the lung.

The rarity of certain types of pulmonary tumors in animals may be related, in part, to various genetic factors. Such conclusions are supported by the work of Heston and others.[44,114-117] Many mammals, birds, and reptiles are exposed to atmospheric

pollutants but have neither an increased incidence of lung cancer nor any obvious urban-rural variation in the frequency of pulmonary neoplasia.[118-131] Atmospheric conditions and pollutants may considerably influence an individual's susceptibility to various pulmonary disorders, such as chronic bronchitis and pneumonia,[132,133] and these factors may, in turn, facilitate the development of lung tumors in humans already exposed to the potential carcinogens in cigarette smoke. The absence of an urban-rural variation in the incidence of lung tumors in animals[58,59,132,133] may be related to the fact that animals are not directly exposed to cigarette smoke, or at least not in a manner similar to humans.

Different factors predispose an individual to developing various types of tumors. These include chronic inflammation, infection, and irritation. The circumstances under which a number of the tumors in this study developed strongly suggest that similar factors can also initiate the tumorous state in animals as well as humans, e.g., chronic ulcers in a stilt's wing (SDZ-8953) or on a pronghorn's eyelid (SDZ-8957) associated with squamous-cell carcinomas; herpetic intranuclear epithelial inclusions associated with a pseudolymphoma in an elephant (SDZ-8085); and the fibrosarcoma of the femur in a maned wolf (SDZ-10282).

The biologic behavior and, occasionally, the histopathologic patterns of the captive wild animals' tumors in this study were often similar to their domestic and human counterparts. For example, many of the animal tumors were found in older age groups, as they are in humans. Multiple primary tumors, not uncommon in domestic canine species, were seen in a captive wild dog (SDZ-11204). Secondary pulmonary tumors were more frequent than primary neoplasms, a finding consistent with most comparative tumor studies. Scirrhous lung tumors, common in cattle, were found in a captive wild artiodactyl (SDZ-7823), and subepithelial glandular tumors, not infrequently seen in domestic cats, were noted in a clouded leopard (SDZ-4795).

There are only several known animal models for the study of the predominant forms of human lung cancer.[134] Moreover, spontaneous squamous-cell carcinomas regularly occur in the wild European hamster, but their frequency compared to that in humans is quite low.[3,135] In addition, only one oat-cell carcinoma has been reported in any animal,[3] and other less common tumors, such as the pulmonary carcinoid, are rarely found in any species but humans.[3,24]

Vertebrates other than humans are generally thought to have a low incidence of pulmonary neoplasia. Although in most cases this is probably true, many of the animals described in this study may have died in the middle of their potential life span. Human pulmonary carcinomas, however, commonly have a peak incidence near the end of the natural life span. Thus, the true incidence of animal lung tumors may actually be higher than is currently appreciated. Still, over 2,000 neoplasms from more than 13,000 autopsies were reviewed in the present study, and among all of these there was not one primary pulmonary tumor of the type commonly associated with smoking in humans.

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