IN THE UNITED STATES DISTRICT COURT

FOR THE NORTHERN DISTRICT OF OHIO

WESTERN DIVISION

MARY A. DENDINGER, et al. and ETTA M. WALLACE, et al.,	:	
Plaintiffs,	:	
vs.	:	Case No. C87-7117
CHRYSLER PLASTIC PRODUCTS CORPORATION, et al.,	:	(Hon. Nicholas J. Walinski)
Defendants.	:	

Deposition of RALPH MICHAEL KELLY, M.D., a witness of lawful age, taken on behalf of the defendants under the Federal Rules of Civil Procedure, in the above-entitled cause, wherein Mary A. Dendinger, et al., are the plaintiffs, and Chrysler Plastic Products Corporation, et al., are the defendants, pending in United States District Court, Northern District of Ohio, Western Division, before Luke T. Lavin, Registered Professional Reporter, a Notary Public in and for the State of Ohio, at the offices of the deponent, Suite 106, 2450 Kipling Avenue, Cincinnati, Ohio, at 10:05 o'clock a.m., on Friday, May 6, 1988.

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would have records in that kind of detail, I don't know. We haven't pursued that at this point.

Q. You don't have this information down?

A. No. That doesn't mean we'll necessarily pursue it, but we don't have that at this point. This would be a very interesting thing to pursue to see if you're saying less than .5, do you know from what he did on that particular day at this time,what his relative time frame spent in those areas where exposure can occur, did he go up on any of the upper platforms where you would expect it to be higher, or, in fact, did he just walk in and out, grab samples and go off and do a little bit of color matching.

Whether they could retrieve those records or not, I don't know. At this point we haven't asked them to.

MR. BUNDA: Can we go off the record for a second?

(Whereupon, a discussion was held off the record.)

MR. BUNDA: Kirk, I'm hearing from your expert that he needs or would like additional information in order to form an opinion. Now, my understanding was that when he was presented for a deposition

today that you had all the information you needed from the series of depositions of my company people, and that once he had that information, he would be able to form opinions.

I'm hearing testimony that he isn't prepared at this point to testify about the exposure that both of these gentlemen suffered, and I'm not going to go through this series of depositions unless he has that information.

I don't want to waste your time, and I don't want to come back and do this again.

Now, are you finished with preparing him and supplying him with the information, or do you have information in the future that you're going to get and give to him?

MR. DELLI BOVI: Mr. Todd's testifying today based on the information and the evidence that has been uncovered to date in this case. Whether or not additional information and evidence will

be uncovered between the present time and the time of the trial, I do not know.

Consequently, I can't tell nor can Mr. Todd tell you what effect, if any, the receipt of additional information will have on his opinions.

Mr. Todd has formulated opinions based on the evidence and the information provided to him today on his education and his background and his experience, and he's prepared to offer those opinions to you today. Obviously, the lack of information in certain areas causes everybody to desire additional information or evidence which may have bearing on the ultimate issues in this case, the opinion of the experts in this case.

Whether or not that information will ever be uncovered or discovered or provided at this point, I don't know. We're in a continual quest to receive and obtain that information, so Mr. Todd is here to give you the opinions that he has based on what

he has received to date, and based upon his knowledge as it exists at the present time.

What the future will bring, I have no idea.

MR. BUNDA: You're presenting me with a moving target. I'm here to get his information, but I don't want to go back and think that I have his opinions when he's going to be getting additional information.

My understanding from the judge is that discovery is closed in this case and that we're now in the process of deposing the experts for the purpose of presenting this case to the jury at the trial of the causation issue, so I'm going to move to exclude any testimony on information that you may get in the future and provide to him which may affect or change his opinion.

MR. DELLI BOVI: Is it your understanding, Mr. Bunda, then, that discovery is closed as to both sides?

MR. BUNDA: It's my understanding that the continuation of the trial was with the understanding that we're permitted to depose the experts, but that we're not going to engage in additional discovery of information from Chrysler. We're here to get the opinions of experts.

MR. DELLI BOVI: Well, I didn't hear the judge say anything at the pretrial that indicated that I was prohibited or that the Plaintiffs were prohibited in meeting with anyone from Chrysler or obtaining any additional information from Chrysler, and if there is something that occurred at the pretrial or is contained in the journal entry reflecting what occurred during that pretrial that bars me from doing that, I certainly strenuously object to it.

MR. MEYER: Just have the record reflect our intention to join in Bob's motion to strike the information

gained at a later date.

MR. BUNDA: Well, perhaps this is a matter we should best bring to the attention of the Court. Since Mr. Todd is here, I'm going to continue asking him questions.

MR. BAMMAN: Bob, what are we going to do about lunch?

(Whereupon, a discussion was held off the record.)

BY MR. BUNDA:

Q We're back on the record. Mr. Todd, would you agree with me that the exposure information that was obtained at the Chyrsler plant on vinyl chloride would be more accurate than extrapolations from, for example, residual vinyl chloride measurements in the resin on estimates by employees of exposure to dust and such things?

A With the qualification, it depends on how in depth the data is from which you can make true estimates. If you have a spot sample here and a spot sample there with no documentation of what went on, then about all you can say is, "I've got a couple numbers."

So with certain reservations in here, that is

correct. In practice, it may or may not be.

Q. You've seen the documentation that was attached to the studies that were done. Is that acceptable?

A. I saw the documents. I don't recall there being that much documentation. As I recall, we did a survey and analyzed the numbers from people or areas that we monitored, and that's not what I call very complete documentation.

Q. What would you like to have in order for it to be complete?

A. Well, what I would like to have, obviously, is an area-by-area evaluation of where you are and what contributes to exposure rather than, "I came in such and such a date. I took a few samples, and I sampled right out by the door, and here's the sample. I'll see you six months from now," or whatever, and more importantly, what would have been far more useful, of course, is data going all the way to the beginning when the operation -- took it over from Airco, 1968, roughly.

Q. Have you formed an opinion about the exposures at the plant to vinyl chloride?

A. In a general sense.

Q. Give me your opinion, please?

A Well, my opinion at this point, of course, is that pre '74, residual levels were much higher than they were post '74, again, from the documentation, my own experience provided.

I know there was a crash program at this point in time to reduce residual levels as well as alert users of this to the potential probelms that might be inherent in it, and that varied from supplier to supplier, but nevertheless, in general, most people using it became aware some time in '74, certainly before the final standard went into motion, because it impacted on them.

Based on what I know about the basic engineering controls in that plant and the off-gassing that I would anticipate, I think you can depict in a qualitative sense what your potential maximum exposures were, and that would be a function of what they did, what the ventilation was in a given area, and how much material it handled, and what their proximity was to the source of the VC emissions themselves, and the closer the source, the more their exposure was; the more remote they were from them, the less they were; and you can see that as well in the production area, and particularly where the individuals went in and out, you would expect to have less exposure,

not necessarily on a minute-by-minute basis, but other than on a daily basis.

Q. Have you examined these exposures from Mr. Wallace and Mr. Dendinger?

A. In qualitative numbers, no. I would expect, however, that you could be looking at levels in those operations in the order of magnitude somewhere between 25 and 100 ppm. I would not expect concentrations higher than that, okay, and the reason I wouldn't is because of the intermittent nature of some of the critical tasks which they do, in addition to which, the data doesn't suggest that the off-gassing concentrations, even though they may have heated or dissolved the material, should have given you peaks above and beyond that concentration.

Now, that's looking at vinyl chloride alone. Keep in mind that their exposure from a health risk standpoint is not telescoped into vinyl chloride monomer in the gaseous phase, period.

Q. Tell me about the other exposures.

A. Well, you would have a number of other exposures, some of which are directly related to PVC and some of which are not. In the case of the dust, I've already gone into that.

Now, keep in mind, when we talk about a parts per million in air, we're talking about a weight and volume relationship, not a weight and weight relationship as we are in the case of residual monomer, so if you inhaled a residual ppm in dust, that isn't the same as an equivalent amount in the airborne concentration.

Above and beyond that, you have the addition of other contamination, whether we're talking in the Ink Room or in the mixed solvents, other dusts, your pigments, your other components. The interaction of those created all sorts of possibilities. In fact, they may not be simply additive. They may have synergistic reaction. You might get a material that is a low level carcinogen, which now, because of the other materials being present, you now exceed the biological threshold.

That's why I also said earlier, just looking at vinyl chloride, telling me, "Here's a number," even assuming that is a true representative sample, doesn't totally depict what the potential health risk is because we have the possibility of the interaction of all weird scenarios in the real world circumstances in the planning of that plant.

Q What are some of the other exposures that are present or were present at the plant?

A Again, I just covered things from the pigment material which could be inorganic to organic. I'm speaking now of the inks when I say "pigments." Obviously, your plasticizer and other materials added to the PVC and other resin as it comes in; some of the other resins; above and beyond that, of course, the PVC itself, and, of course, the solvent.

You know, we know from a physiological standpoint what specific materials do in man and animals. When you start intermixing materials, our knowledge becomes a little more gray. Consequently, most industrial hygienists will say, "Well, I don't recommend you push the TLV of this, of that, and the other thing," because even though we may not necessarily affect the same target organ, we don't know what the interactions might necessarily be, and therefore, on the side of coercion, drop your numbers accordingly, and where you can, to the lowest extent that's feasible within the economic considerations that you have to work with and what is available in terms of technology.

Q You know the type of cancers that we're talking about in this case. Do you know of a synergistic reaction between vinyl chloride and any of the other chemicals that are out at the plant that would result in these type of

cancers?

A. No, only because I don't think it's ever been studied, and if it has, it's a well disguised study.

Q. Have you done a review on any literature of any chemicals used in the plant to determine whether they alone can cause these cancers?

A. There's probably a few materials; again, we don't have, perhaps at this point, the access to the nature of all the other materials that are used there, though I have a feeling getting that information wouldn't be too difficult to do in the foreseeable future, and, again, you'd like to know what they are in more specific terms.

I don't know what, for example, the ink formulations are. I don't know what specific plasticizer they use. I don't know what fillers and so forth that they also use in that plant.

Q. And you would like to have that information before you form a conclusion?

A. Well, it might be useful, and I said, "might," because, again, you wouldn't know until you get that information, and then, again, go back and say, "What do we know"in response to the very question that you posed.

Supposing one of those other components there

does interact, and now you have almost a classical asbestos-cigarette smoking combination where either -- which either, in itself, are relatively low level carcinogens, but together synergistically react to form a very potent mixture, and now instead of getting a simple addition reaction, you get a multiplication, so that's a possibility.

Q. And as we sit here today with the information available to you, you don't know about those synergistic reactions; is that right?

A. No, simply because you'd have to dig further, assuming Chrysler is willing to, one, give you the information, and then, two, the suppliers, really giving them adequate information instead of this is a proprietary secret and we're selling you plasticizer agents and we're not going to tell you anything about it. That's always a potential problem.

Q. And as we sit here today, you cannot say whether any of the other plasticizers or pigments or inks or things may or may not have attributed to this or caused this cancer?

A. In an absolute sense, you can't; however, I haven't seen anything specifically popping out of the literature that says that a specific plasticizer or pigment is

Q Is what?

A Is carcinogenic to the lower G.I. tract and/or, in this case, the gland tissue. That doesn't mean it isn't, but I'm not aware of it.

Q And you haven't done a study of the literature on that issue with regard to vinyl chloride either?

A Well, you'd have to know the specific compound, first of all, and therefore, you have to have a willingness for them to cooperate and provide that information so you can then do what you're suggesting.

Q But listen to my question. You have not done such a study with regard to those questions and those cancers, either.

A That's correct, simply because you'd have to know what they are at this point. We don't have that information in hand.

Q So your testimony is addressed to the exposure level to vinyl chloride and polyvinyl chloride and not what those exposure levels mean in terms of causing disease?

A That's correct. That's correct. Again to repeat what I said earlier, I wasn't asked to make a

medical judgment on that.

Q. Let's address now the figures of -- or your estimate of an exposure to vinyl chloride monomer of 25 to 100 parts per million with regard to those two specific gentlemen, Mr. Dendinger and Mr. Wallace.

What's the basis for your opinion on that?

A. Again, I'm using some of the -- both the B. F. Goodrich estimates from their field studies and calculations, as well as some of my own, which admittedly, only go back to early '74, but I'm assuming that early '74 is probably a good reflection back to '68.

Q. When you mention you're using the B. F. Goodrich studies, you're using the mathmatical computations which they made; is that right?

A. Well, if I understand, what they put out is not just mathmatical, but really a field study in which they back calculate mathmatically to derive with some statistical confidence air concentrations that you anticipate as a function of the residual monomer levels.

Q. Well, Mr. Todd, with all due respect, wasn't their study addressed to the issue of what residual vinyl chloride monomer level could be achieved in the resin with the ability to also say that the action level wouldn't be

exceeded if you had that residual level? Do you understand what I'm saying?

A. Well, their ultimate goal was to try and determine, and I suspect from a regulatory standpoint as well, for in-house purposes, what level of residual monomer would they project from both their field experience and calculations would not result in exposures over the proposed OSHA standard of one ppm, and what they were doing was putting together their field data to arrive at some sort of statistical confidence as to their projections, presumably so they could use them in house, but perhaps they also disseminated them to some of their customers.

Q. And you're taking that information and extrapolating it to the higher level of residual vinyl chloride monomer level you say existed before 1974?

A. Yes, I'm doing that plus again using some of my own experience from that early '74 era, in general, warehousing and related facilities aside from production.

You know, when you talk about 25 and 15 and 100 ppm in some of the production areas, when spills occur or they had the blow out reactors and so forth, the numbers would almost get astronomical. Of course back in the early '74 era on, they had some pretty sophisticated respiratory

equipment on those people.

In the warehouse areas, I think the assumption was made that the final standard would probably be around 25 ppm; therefore, if you didn't have a huge multiple of that, it wasn't really a problem.

Q. I'm focusing now just on the Goodrich study you mentioned. You have not done any additional modeling of your own with regard to whether that study applies to higher levels, have you?

A. They presently did it with higher levels as well as the lower levels, if I understand the data point.

Q. Have you done any study with regard to how long it took to shift, for example, the Goodrich resin from the Goodrich plant to Chrysler, or how long it was in the warehouse?

A. Well, again, I'm going to repeat their responses, and I say "their," I'm talking about Goodrich and everybody else's, so basically they say they don't know, it could have been a few days; it could have been a month; it could have been anything in between.

Q. Would you need that information in order to arrive at an accurate estimate?

A. Well, if I understood their degassing,

barring the material being extremely porous, while it would reduce in months, it wouldn't be 10 percent, okay. More importantly, they've already admitted they have no records one way or the other to indicate whether it took two days or a week or a month.

In most cases, they agree they took and shipped it as quickly as they could for, obviously, economic gain, plus particularly in the '73-74 era for the sheer demand versus the amount available in the world market.

Q. That's presuming, in fact, that any came from Goodrich or any of the Defendants at all.

A. I'm not talking Goodrich, per se. I don't know who supplied exactly how much in a specific year -- maybe in a specific year you do, but on a specific day. You might as well ask me who supplied the material on July 31 of 1974, and the answer is any or all of them except somebody who said, "We sold to them in 1979," in which case they, obviously, couldn't have.

Q. You've already testified that in your experience there was a shortage of PVC material in '73 and '74.

A. Yes, that's correct.

Q. How did you get that information?

A. Well, first of all, I worked for the oil industry, so I was aware of the basic shortage of feed stock to petrochemical plants in general, but more importantly, that is also verified in a number of the depositions from various individuals who were deposed from Diamond Shamrock and Goodrich and Union Carbide and so forth, and there's continuity all the way across the board that, yeah, we agreed, in that time frame; and again, that's even consistent with some data and input from, interestingly enough, Goodrich in the Celanese cases.

Q. Isn't it also true that the method of a manufacturer can affect the final effect of the vinyl chloride level in the final polymer?

A. That's correct. It would appear that certain processes leave far greater amounts than other processes do.

Q. And catalysts in the polymerization also affect the vinyl chloride level?

A. In the general sense.

Q. Are you also familiar with the fact that certain foreign polymers contain higher levels of residual vinyl chloride monomers?

A. The question is yes and no. I've seen

suggestions that may be so, but I haven't seen any real hard data to confirm that.

Q. But you expect, for example, resin made in Mexico to have a higher or lower level?

A. It could, depending upon who made it. You know, for all I know, B. F. Goodrich had a plant there and they made it in their Mexico plant. If it was the same company and their criteria and process were the same, it may very well have the same amounts, so it depends.

MR. BUNDA: I'll move to strike the answer on the basis that I don't think it was responsive to my question.

Q. Are you familiar with the fact that Goodrich or that Chrysler during '73 and '74 was buying Mexican resin?

MR. DELLI BOVI: I'm going to object to the question. Go ahead and answer.

A. I'm not sure that I'm that familiar that -- I do recall some coming from overseas, but whether it was from Mexico or Europe, I don't recall.

Q. Are you familiar with the fact that the residual vinyl chloride -- strike that.

Q. Are you familiar with the fact that the vinyl chloride readings taken in the head space of the Chrysler storage bins was reflected in some of the testing in '73 and '74?

A. I don't recall any testing in '73.

Q. All right, in '74?

A. Restate that question again?

Q. Are you familiar with the fact that the vinyl chloride levels were reflected in some of the head space readings taken in '74?

A. I'm not sure I understand the question. I understand what you're getting at by "head space readings," but reflected where?

Q. In the studies that were done.

A. You're losing me, I'm sorry. I don't quite understand your question.

Q. Are you familiar with the fact that vinyl chloride readings were done in the Chrysler plant?

A. Sometime in '74, '75 on.

Q. The answer is yes?

A. The answer is yes.

Q. You are familiar with the fact that the vinyl chloride gasses were measured not only from personal

exposure, but also in head spaces?

A. I believe that is correct in the silos or rail cars, and I've forgotten now which, and I say I believe that, and I'm saying that from distant recollection.

Q. You're not familiar with having reviewed that recently?

A. No, it's been quite some time since I've seen it and --

Q. Are you familiar --

A. -- andit's a little bit fuzzy on my part.

Q. Are you familiar with the fact that some of those head spaces reflected the vinyl chloride gas which was given off and that resin was identified as being Mexican?

A. Again, I don't specifically recall it. I'd have to go back and look at the data. That data doesn't come up in my mind.

Q. And you're not -- do you have any recollection as to whether those head space readings were higher or lower than domestic resin?

A. Again, I'd have to go back and look at the data. It doesn't come to mind.

Q. That has no relevance to you in your analysis?

A. It would have relevance, but I just don't recall

the data you're referring to. That's what I'm saying.

Q And if, in fact, the Mexican resin was higher, that would have some bearing on the exposure levels, according to your analysis; is that right?

A Yes, absolutely, if you're degassing more readily and you've obviously got a greater potential content there, and it's reflected head space sample, yes, you would expect obviously a greater occupational exposure as you actually utilized that resin in the process areas.

Q And am I correct in presuming that in your opinion and in your analysis, you did not break down the exposures of the two gentlemen involved in this case to the particular resins of each of the Defendants; do you understand?

A How could you? Stop and think about it. That's like asking somebody, "Whose resin did you use on Monday; whose did you use on Tuesday, and how much did you use each day?"

You have enough difficulty trying to figure out how much was used each day without somebody trying to remember whether they used Company 1, Company 2, and/or Company 3's on Monday and so forth and so on. So you really couldn't do that if you wanted to. I'm sure they

couldn't provide those kinds of records.

Q. And you did not make an analysis about the comparitive exposures caused by the Defendants' resins versus, for example, the Mexican resins?

A. I assume the Mexican resin is also from a Defendant.

Q. And why do you make that assumption?

A. Well, I'm assuming that anybody who supplied the resin would be automatically a Defendant. Now, why would one resin -- unless you could prove that one resin was totally VC free, why would they be excluded, barring, you know, being sold at one time other than when these gentlemen were employed by the company.

Q. How did you come to the 25 to 100 parts per million figure?

A. Again, I used the B. F. Goodrich and also compared it with my own experience during that same general time frame. This is before the more rigid stripping and so forth procedures were instituted, as well as ventilation modifications and what have you.

Q. I'm sorry. Showing you Defendant's Exhibit 1, is this the B. F. Goodrich document that you're referring to?

A. I have a feeling there's another document besides this. This isn't the only document. There's another one, and I have a feeling the other one I'm referring to postdates this.

Q. Did you use this document in assisting you to arrive at that conclusion of 25 to 100 parts per million?

A. This document was reviewed and there's a follow-up document, and I don't know whether the other -- well, I'm sure it's in this pile here somewhere.

Q. Listen to my question. My question was not whether or not you reviewed it. My question was did you use that in formulating your decision on your opinion?

A. I may have in part. I'm sure I looked at it.

Q. What part are you referring to of that document which assisted you in arriving at your opinion?

A. Well, this is, again, a physical modeling system where they're trying to project under less than severe conditions whether you're going to exceed the action limit of a half a part per million based on the residual vinyl chloride content, and they're taking into account various factors, okay, in their modeling system, and they put together documentation in terms of tables and graphs and so forth, but this isn't a specific one I'm

referring to here.

Again, they make projections here based on temperature, air turnover and so forth as to how much airborne vinyl chloride monomer they're going to anticipate finding based on the actual content, measured content of residual vinyl chloride monomer.

Q. Okay, but again that is a mathmatical --

A. There's one that postdates this in which I've got actual measured air concentrations, and in part, this is a very useful document because it does give some measurements and estimates of their projected air concentrations as a function of temperature and other variables and the actual residual levels in the PVC itself, so this is the first of at least two documents in making those projections.

Q. Downward?

A. Making those projections downward.

Q. Well, my question -- I'll strike that question.

My question to you is what specifically did you derive from this document which led you to your conclusion of 25 to 100 parts per million was the probable exposure at the Chrysler plant for the Plaintiffs?

A. Well, to answer your question, I didn't rely

on this document alone for making those conclusions. The information in here is useful as a prelude to one which followed.

Q. I don't understand that. How do you mean as a "prelude?"

A. Well, this was an experiment in an attempt to derive the concentration -- or to arrive at a concentration of residual VCM, which is, in theory, if you sent it out, would always meet the action limits, okay, and they project a number of data and graphs to support their theory; but above and beyond that, in a few head space samples here and there, there's not a whole lot of data to support conclusions as to what actual field concentrations under plant use conditions would be based on vinyl chloride residuals from this document alone.

So, you know, it's useful, but it's not a complete document in itself from which you can draw a conclusion. It has a useful piece of information in it.

Q. That's what I'm asking. How did you use this document to arrive at your opinion?

A. Again, they're going into the various variables that will affect the release of vinyl chloride, so it's -- it's background information. You can't use this to

mathmatically extrapolate anything because it makes certain assumptions which aren't necessarily confirmed by field data, so it was a first step.

Q. Where is the other document that you relied on for Goodrich?

A. Well, that's a good question, in this big heap of paper. Let's see if I can find it. That was what we started looking for before, and we quit before we finished.

(Whereupon, a discussion was held off the record.)

A. Maybe I'll find it over our lunch break. Maybe it's in the B. F. Goodrich pile. I'm pretty sure it's their study, which is a follow up of this one here, and I think it goes a year or two later.

MR. DELLI BOVI: Here's some of the Goodrich documents. I'm not sure if it's there or not, but that's Goodrich stuff.

THE WITNESS: I'm pretty sure it's a Goodrich document. This might possibly be it. This isn't -- this is the follow up study, but it's not the one.

There's two of them in sequence,

but there's a later one than that. This is this one here that we're referring to.

BY MR. BUNDA:

Q That's Exhibit 1?

A Yeah, that's Exhibit 1 followed by Exhibit 7, but there's a later one than Exhibit 7.

Well, you say Exhibit 3. There's an Exhibit 7 sticker on it, but it's also labeled Goodrich on it.

Q We have to do this for the record, Mr. Todd. It's going to confuse things. I'm just trying to see -- where is Exhibit 1?

A It's a Goodrich document. They're both Goodrich documents.

Q Can you find the record that you're referring to?

MR. DELLI BOVI: I don't know if it's here. You looked through it once.

THE WITNESS: If you've got the Wheeler depo, I could probably find reference to it in there, but I know we've got it, and, unfortunately, maybe we should have red tagged the darn thing.

(Whereupon, a brief recess was taken.)

BY MR. BUNDA:

Q. Mr. Todd, as I understand it, you're having problems finding that specific record in your documents; is that right?

A. That's correct. We found reference to it in one of the depositions, but now we have got to find it.

Q. Well, without looking at the document, can you tell me how you used it?

A. Well, again, I was using it for comparison with my own field experience.

Hold on a second. It doesn't look like it. If it is, I can't read it. I would say there's some missing things there. We'll have to go back and --

Q. Well, Mr. Todd, tell me about your field experience, then.

A. Well, I guess that goes back to some time in very early '74, I would guess, January or February of '74. We were originally retained by Air Product PVC Manufacturing. I think they have either two or maybe three PVC plants.

Q. PVC Manufacturing?

A. PVC Manufacturing. We also worked with SPI that same year as they were trying to lay groundwork for response to OSHA and their own proposals for a

practical, realistic vinyl standard.

Q. When you say "we," you're talking about your company?

A. Stewart-Todd Associates. Dr. Stewart was doing the medical aspects, and I was doing the industrial hygiene in conjunction with Air Products and at their facility.

Q. What did you do for Air Products?

A. Basically set about in getting air monitoring programming going, doing data evaluation, and at the same time giving some alternatives where we could for solutions to some of their exposure problems, short and long term.

Q. Did Air Products have any PVC fabricating facilities?

A. Not that I'm aware of.

Q. You weren't involved in measuring fabricating facilities?

A. Not for them, no. I've been in a number of fabricating plants since, but not for Air Products. Most of my experience, however, in fabricating plants probably is post '78.

Q. How did you get experience in that?

A. Particular demand from the client to do a

fabricating plant of one type or another, pipe, fabric, whatever.

Q. Were you measuring for vinyl chloride or all types of exposure?

A. Depends upon the client. Some were strictly vinyl chloride; some were mixed exposure, depending on what their request was.

Q. Did you do a report for Air Products?

A. We did a series of reports.

Q. Do you still have copies of those?

A. Unfortunately, that's too many years ago, no. They're gone. I don't know whether we returned them to them, or they got to the point where we asked them if they wanted them and they said they didn't, so now we don't have them in our files any longer.

Q. Who were you working with at Air Products?

A. A guy by the name of John Novac, and Novac is no longer with them, and there's another gentleman whose name I can't recall, but I may be able to go back and find out.

He was in their upper management. Novac was their brand new industrial hygienist at that point. He had just been hired.

Q. What was your contact with the SPI?

A. Air Product was a member of SPI. Air Product was looking for third-party data and reports from which they could support a final standard. This is back during the early days of the recommended interim standard, and then the interim standard itself.

Q. Did you have any involvement in assisting SPI to take a position on the proposed government regulations?

A. The answer is yes and no. We provided input which went into SPI. I don't think it was necessarily accepted wholeheartedly.

Q. What was your input?

A. We told them we believed that by the end of 1975, in a practical sense, they could attain and live with a 10 ppm standard. The impression conveyed back to us or the attorneys was they were really looking for some higher number, so subsequently, I doubt that our guidance went far beyond -- it became an academic point because before that discussion got very hot and heavy, OSHA came along and promulgated the one ppm, at which point it became an academic discussion.

Q. Did you have any involvement with the OSHA hearings that were held in 1974?

A. No.

Q. Have you ever reviewed any portion of the transcript of those hearings or any documents submitted?

A. There was a number of documents in there that were presented at the OSHA hearings or to show OSHA on behalf of the various Plaintiffs and so forth.

Q. When you say, in there," you're talking about the materials the Plaintiff's attorney provided to you?

A. That's correct.

Q. These were materials he selected out of those hearings?

A. These are materials he received from discovery and collected out.

Q. Well, you don't know whether they were received from discovery or not. You were provided with those materials by the attorney.

A. Well, I think the answer is yes and no,because the Interrogatories requesting them were attached to those, so it would appear as though they were in response to discovery requests.

Q. Did you see documents from the OSHA hearing other than attached to those responses?

A. Not specifically.

Q. You've never reviewed the transcript of the

hearings or the documents produced recently, have you?

A. Recent vinyl chloride hearings?

Q. No, I'm talking about the 1974 hearings, you haven't been through those recently?

A. I'm sorry, I misunderstood you. The answer is no, I have not reviewed the entire 1974 hearings input. I'm sure there was a lot from labor, from industry, from academia, et cetera, et cetera, but, no, that would be a voluminous task to go through it. I wasn't asked to and I didn't do it.

Q. Have you seen any information in there concerning exposure levels in fabricating plants in 1974?

A. Are you talking about in the OSHA hearing now?

Q. Yes, sir.

A. Again, I haven't read the whole hearing review in detail to indicate specifically -- there's a few attachments in there that talk about fabricating plant data, sometime perhaps as early as late '74. I think that's your earlier data. I think basically that that data was submitted in support of attempting to get exemption so that the fabricating plants would not come under the standard.

Q. You haven't seen any fabricating plant data from early 1974?

from our site visit than I did the first one which was rather rushed and under a different set of circumstances. I would probably want to reflect on that and ask a few questions in order to come up with the sort of figure that you're looking for.

Q. What would you reflect on and what kind of questions would you want to ask?

A. Well, as you know, there is a limited amount of data in the plant, some of which isn't even on Wallace himself. My real questions come down to understanding a little bit better what are your sources of emissions, and where are they in relationship to them, and then if there is any monitoring done, where were they on those specific days, because, again, that would give you at least a retrospective index of what their TWA is, so I assume from the data I saw those all represented long term or reasonably long term exposures.

I didn't see any of the Chrysler data, again if my memory is correct, which represented eight-hour samplings, and that was one of the problems I had with some of the data was that the exposure period, you sampled for a guy from X to Y, how representative is that of the full shift, or is that the maximum, is that the average, or is that even

typical.

Therein lies part of the problem with TWA definition at this point.

Q. What information could you get to allow you to determine in your own mind what the TWA exposure was?

A. I'd have to basically understand a little bit better than I currently do what they did and how long they spent in those areas and how much material they handled specifically in those areas, were processed through those areas.

Q. How would you get that information?

A. Well, hopefully, Chrysler will provide it. They were far more helpful today than they were last October.

Q. Tell me about the October visit. What did you do?

A. You were along, correct?

Q. No.

A. No, okay. I did a preliminary walk-through survey of the Ink Rooms and the bag storage area, and in addition to which, took a look at the engineering controls, one,to get an idea of what they were and how much we wanted to ask regarding them; and, two, to try and get an estimate

of when they might have been put in based on appearances, so that's basically what occurred in October.

Q What sort of conclusions did you form after you visited the plant in October?

A Conclusions, at least in the Ink Room, there had been considerable amount of ventilation added sometime in the 1970's, and while it may not be the Cadillac of local exhaust, fairly good.

The impression pervaded in the answers indicated that most of that was, in fact, installed mid '70's or after. I wasn't aware at that time, I believe, that they had an OSHA citation on the solvent, so part of that's related to that.

My impression in looking at the data and asking questions at that point is there had been some dramatic decreases in vinyl chloride levels and product received as well as anything that they could see in terms of the air monitoring that they did. That's kind of first impressions.

Q There were dramatic decreases in the product they received and the air monitoring because of the ventilation. When did that decrease occur?

A Well, I don't think -- I don't think I said the second part. I said there was dramatic changes done

in the engineering controls, the ventilation. They indicated there was dramatic increase in the amount of residual monomer present, in product received.

Q Increases or decreases?

A. Decreases.

Q What is your understanding about when these decreases occurred in the residual vinyl chloride monomer?

A. Are you asking me in terms of their input or my impression from all the documents?

Q Well, let's take it one at a time. What did you learn from them?

A. My impression from them initially was sometime in late '70's, they had gotten down to almost where they were currently, okay. My impression from the documentation isn't a great deal different excpet that if you look at their numbers, it would say that occasionally there's still a lot getting by here and there, which will show any residuals, but by and large on the average, even those reflect dramatic reductions.

Q Did you see where Mr. Wallace and Mr. Dendinger worked when you viewed the plant in October?

A. I saw the Ink Room and related areas. The production areas where Dendinger worked which I found out

somewhat after the fact, I didn't get a look at until today because he worked there as well as in the Ink Room.

Q. What is your understanding concerning where Mr. Dendinger worked?

A. I'd call that, for lack of any better description, a coating operation, cloth coating operation.

Q. What did he do?

A. I gather he was an equipment operator, and I'm not sure where exactly he worked on that line, or if, in fact, he alternated from one day to another, but it's a typical cutting line starting with your raw substrate which is going to be coated being coated at the first step, going through a series of process steps, dried, and ultimately at the other end being rolled up as a finished fabric.

Q. What is your understanding with regard to what Mr. Wallace did?

A. My understanding is basically he worked primarily in the -- in or around the Ink Room in his more senior years as a color matcher; in his more junior years doing the nuts-and-bolts type operations from blending of pigments and clears and similar solutions.

Q. How did Mr. Dendinger get this exposure to vinyl chloride, in your understanding?

A My understanding is that he mixed the basic components for the coating, and it was coated directly on the material, and in addition to the proximity to the storage, you actually have a blending operation as well as a coating operation all at the one end. I would expect far greater exposure than if you worked at the other end where they're just basically rolling up the vinyl-coated fabric, or basically, after passing through the dryer and the elevated temperature, you would expect little or nothing present.

Now, that doesn't mean zero, but you would expect relatively little as you go a distance from the original source, and also having passed through a high temperature hot air dryer, it should have almost completely degassed.

Q The Ink Room is apart from the mixer and the storage area?

A Physically separated by block walls from most of the plant except for the doors going in and out.

Q How did Mr. Wallace get his exposure to vinyl chloride?

A Well, again, you're using bagged PVC for making up initial clear materials, meaning your PVC and solvents,

and then, of course, you're adding pigments with agitation, and so you've got your potential during the actual bagged up thing; you got your exothermal reaction, your heated solutions as you actively agitate it in a large tank, and then if you follow it down to the feed and other drums where you're diluting it up or making it up with pigments to specific colors, so any and all those sources, it loses the material due to either the exothermal heat reaction or due to the fact that it's going off with the solvent vapors.

Q. Do you have any understanding as to what type of polyvinyl chloride was used in that operation by Mr. Wallace?

A. I did at one time, and I've forgotten the details of that. When you say "type," I presume you mean was it an emulsion or suspension manufactured process --

Q. Yes, sir.

A. -- product.

Q. Yes, sir.

A. If I understand, I believe, from Wheeler's deposition, the impression I get is it's an emulsion or modified emulsion process, and I'm saying that from recollection. It seems to me he cited those type applications, products of that type.

Q. Does that have a significance to you with regard to the contents of residual vinyl chloride monomer?

A. Well, yes, in a general sort of way.

Q. It's lower, isn't it?

A. Pardon?

Q. It's lower, isn't it, in that type?

A. No, I have a feeling we're talking about two different materials. If you're talking about some of your microsuspensions and so forth, it would be lower. But if I understand Wheeler's data correctly, and his deposition was interpreted correctly, those would be some of the higher ones.

Q. What would Wheeler's deposition have to do with the type of PVC that Mr. Wallace used?

A. Because Wheeler describes basically the four basic processes and what historic data is available on residual monomers. I say "historic," pre-1974 data in addition to which he discusses relative losses that you would anticipate based on the porosity or lack of porosity in those molecules -- or pardon me, in those particles.

Q. Have you talked to the people at Chrysler about the type of PVC they used in the Ink Room?

A. Not me specifically. I think there was some --

Q. I'm sorry, go ahead.

A. I think there was some information on purchases, but unfortunately, I don't think that specifies types.

Q. Well, other than you specifically, do you have any information which indicates what type of PVC was used in the Ink Room?

A. No. That would be useful information to confirm Wheeler's suppositions.

Q. And that would have a bearing on the exposure that Mr. Wallace had?

A. It could. Again, if it were either end of the spectrum, it might change your opinion somewhat.

Q. It might change your opinion?

A. I meant my opinion, yes. I was speaking in the third party there.

Q. And the exposure as a color matcher, that would be less?

A. Yes. Well, if I understand from my insight, the color matcher, he basically goes in and out of the Ink Room, and his job, of course, is to feed back information indicating to the striker, assuming he isn't the striker also, and that fits into the description as well, tell him "Add so much more of this," or, "No, it's fine," as the

case may be, and then review it in terms of color matching, too, what they're specifically trying to match from a former lot or whatever.

So he's going to go in and out of there intermittently, but he doesn't need to be there eight hours a day, and that's what I said earlier when you said they had the data on Wallace, the question that occurred to me is, well, fine, what was he doing at that day, if he was a color matcher at that point and specifically the time in there, it's interesting data for that time frame, but may not totally be applicable before or after, depending on what his job was that day.

Q Did you see where the color matching is done in that plant?

A Yes.

Q It's an office?

A I'd call it a small laboratory for lack of a better term.

Q It's self-contained.

A Well, it's self-contained, but it's not negative pressure. Don't get the impression because it's self-contained that nothing that's out in the plant will move into that area, but I have a feeling, based on where

it is and how it's set up, that it's just general plant contaminants rather than from specific PVC areas.

Q Have you done a report in this case?

A No.

Q Have you been asked to?

A I still would like some more data.

Q Have you been asked to do a report?

A I'm sure at some point I will, and I'd still like some more input before I finalize the report for some of the reasons we've already discussed.

Q Without belaboring what we've already talked about, are there specific data that you would require before a report is prepared?

A Yeah. I think we've already touched on some of those. I don't know whether we touched on all of them or not, but a number of them certainly we have.

Q That's what I'm asking. Other than those that we've already discussed, is there anything else that you need?

A Well, a number have been brought to the forefront. I guess, again, to repeat what's already been said, you'd like to know what the spectrum is of what they've already been exposed to, including what PVC's; two,

you would obviously like to know whether there was a specific type of PVC used in those operations, or whether it really doesn't matter, and, therefore, it could have been any kind.

Obviously, it's important to know when ventilation changes were made and quantitatively how you changed the air to radically cut off the specific source of exposure as a function of time. So far, Chrysler already has basically said, "We put in ventilation in 1970. We put a little bit in. We put some more in in '74. We put in some more in '76."

They haven't quantified what this ventilation was like in terms of actual capacity on the various type units that they did put in.

Again, that will have an impact as well on some of those other things that we discussed.

Q Focusing for a minute on the exposure in 1974, do you have -- and I'm talking now about early 1974, March of '74 --

A Uh-huh.

Q -- do you have any information that the exposure to vinyl chloride resulting from the use of PVC at Chrysler was different at that point in time than it was

in 1968 or any period in between?

A. Well, you're asking me to extrapolate a bit. First of all, it depends on how much data they have in March of '74, okay, and how good the data is.

Assuming that were a good data base, I would expect '68-74 to be equivalent, possible exception, they did make minor modifications in '70, but I would really expect them to have a big impact, if I understand what they did.

Q. And by "they," you're talking about --

A. Chrysler.

Q. Okay. The work that you did with the SPI, did you produce any reports for them?

A. No, the reports we produced were for Air Products, who were feeding through some sort of committee to SPI, and I think what they were looking for was a series of documents from third parties outside of their organizations in providing additional input as to directionally where they could go; and, too, in a practical sense, what could they live with and attain in specific time frames.

Q. Did you ever communicate with anybody directly inside SPI?

A I think the answer, at least from the industrial hygiene standpoint, is no. I can't speak from the medical standpoint, because I'm not positive about that because we were feeding both pieces in. My impression, and that's why I'm pretty sure we didn't communicate directly, SPI was not happy with our suggestion that a lower number could be attained, and, therefore, came back to Air Products and asked us to reassess it, and, no, I don't -- I don't recall that we had a direct pipeline to them.

Q When you say, "A lower number could be attained," you're talking about an exposure level in the manufacturing plant; is that right?

A Yep.

Q That didn't have anything to do with whether or not a lower number could be attained in a fabricating plant?

A Obviously, if you can attain that in a manufacturing plant, you have a better possibility of attaining it in a fabricating plant.

Q Well, I understand that, but listen to my question. Your comments were not being solicited with regard to the exposure in the fabricating plant.

A. No, they were geared to the plants which have more significant problems.

Q. And that being the PVC manufacturing.

A. PVC manufacturing. It wasn't even VC manufacturing. We didn't get involved with VC manufacturing simply because, as I understand from people who did, they had far fewer problems than the PVC manufacturing plant.

Q. Did you have any involvement with the Manufacturing Chemists' Association at this time?

A. No, not directly. The only other involvement I had in terms of trade associations was I was a member of API, and API, obviously, had a particular interest in vinyl chloride because most of the vinyl chloride manufacturers, and speaking now of the monomer, are API members. It's basically a petroleum byproduct.

Q. What was the extent of your involvement, then, through the API?

A. More in keeping abreast as to what's going on in the other two trade associations, as well as to what sort of data was coming out, was in terms of technology and otherwise.

What I'm saying is there was a fairly good interchange between the three trade associations because

there was a lot of overlapping membership.

Q. Have you been receiving copies of the depositions of the company employees that have been deposed?

A. Are you speaking of which company? You mean Chrysler Company employees?

Q. No, I'm talking now about the PVC manufacturers.

A. You're losing me.

Q. Certain depositions have been taken of employees of PVC members who are Defendants in this case? Have you received any of those depositions?

A. Oh, you're talking about former plant managers and so forth?

MR. DELLI BOVI: Well, let me clarify. He has a copy of every deposition that's been transcribed in this case.

A. I'm sorry. I was thinking in terms of nuts-and-bolts operations. Yes, if they were deposed, there's a good chance I have the deposition.

Q. What information have you taken from those depositions to form your opinions?

A. Well, they're all reasonably consistent. What I hear all of them saying is -- well, depending upon their level within the company, many of them, of course, knew

about the Loyola studies and the Maltoni study in 1970 to '72 time frame, and, of course, the MCA study which got sponsored in '73 as well.

Others, of course, were totally unaware until the B. F. Goodrich announcement of angiosarcoma in January of '74 that there was even anything above and beyond the acro-osteolysis problem of the mid '60's. In fact, a few of them didn't even know that.

Consequently, what I hear most of them saying is, "Our monitoring of our employees as well as products prior to January of '74 ranged from nonexistent to extremely minimal," the latter being primarily using a standard explosimeter to make sure that you didn't blow the building down or the reactor and so forth, which for occupational health purposes is better than nothing but very close to it.

Then suddenly in 1974, they're faced with a problem of unknown magnitude, and then begin to react in fashions that they probably could have done several years before, but either didn't know enough or the company wasn't so inclined, or everybody was waiting for somebody else to set the pace. That's my -- my impression of those depositions in a general sort of way.

Q. Given your involvement in this case and the

review of those depositions and the other materials that have been sent to you by the Plaintiff's attorney, do you have an opinion about when the residual vinyl chloride monomer level should have been reduced by the companies?

A. Sure.

Q. Tell me about that.

A. Well, Viola's work was the red light on blinking which said, "Hey, fellows, admittedly this is an animal system, and it's in high concentrations, but if you believe the data, then there may be a problem."

That data was reviewed and discussed, and it wasn't sponsored by U.S.-owned companies, but was certainly available to them, depending on who you listened to, anywhere from the Houston paper in 1970 to a follow-up conference in '71.

At that point, I would have expected two things to occur as a professional: One, for people to go out and begin to get quantitative data, not wait until three or four years later to begin to develop the procedure so they can; and, two, to begin to say what is the potential impact on us if it really turns out to be so, and how do we get the ball rolling so we can find out whether it's real or just an artifact.

But, in fact, that ball didn't roll very far until '73 when the MCA study finally got going, at which point some of the Maltoni studies and so forth were beginning to come out, and still nothing got done until, of course, the B. F. Goodrich announcement, at which point they quickly hired Tabershaw-Coopersmith Associates to do some epidemiological data and see whether the problem is even real, and if it is, the scope of it.

So everything basically sat around for four years and didn't go anywhere, and, so, yes, they could have -- the methods that developed in '74, '75, '76 were methods that could just as easily have been discussed and implemented in 1970-71, so there's a real time gap.

Now, again, I think they're operating in the usual, I'd call it, tunnelvision. You know, this is animal models, and we hit them with high doses, and, therefore, it doesn't mean anything in man because nobody can count the bodies, and I'd go along with that philosophy except that in the real world, you don't have any occupational exposures close to pure vinyl chloride and pure vinyl chloride alone, and, therefore, on the side of caution, a good product stewardship role says do your homework at a reasonable time frame, not when it becomes economically

convenient, and/or the other guy will go along with me and we'll fund it together two or three or four years down the road.

As a matter of fact, in a realistic fashion retrospectively, based on the sheer volume of PVC manufactured, and again, we're talking millions, and by the late '60's, billions of pounds per year, they really should have done the product studies probably a decade before Violas. In the current time sequence, it would be done even before that now.

So you have two time lags, one between the time when the initial signals were given and still nothing occurred for almost four years; and then more importantly, even that was late in a realistic sense. The industry could well have afforded to do those sort of chronic studies perhaps a decade before that.

You're looking at something pointed out in the OSHA hearing, thousands of employees who were exposed to millions of pounds per year of manufacturing, and yes, up until those European studies, nobody had gone beyond a subacute, and the subacute didn't look that complimentary.

Q Your opinion is that on what date they should have began doing studies on long-term exposures?

A Well, without having a year-by-year manufacturing record, as I understand it, PVC went commercial in the late '30's. There was acute studies done around that time frame, and thereafter through the '40's. There was limited subacute studies done in the '50's.

In theory, in the late '50's or early '60's, depending upon the level of growth of that industry, it would have been a time to initiate the long-term studies.

Q I'm sorry, what was the date again?

A I said the late '50's to early '60's. Again, I'm making some extrapolations or estimates, as it were, as to what the growth rate was in terms of yearly production.

Q I don't understand. How do you tie the production to when investigation should begin?

A Well, let's look at it in a realistic sense. You can't say to a manufacturer or industry, "You're producing the first drum of product X. You've got to go out and spend a hundred thousand or a half a million dollars to do your own studies that are necessary to prove what the long-term health ramifications are."

You can when -- you can realistically ask an

industry who is beginning to go commercial, "Hey, go out and do the simply acute things." First of all, they're inexpensive to do, and people need to know are they irritating, are they sensitizers, will they kill you systemically in a relatively short period of time, and those are cheap to do.

When you get to the point where it's obvious that your exposed population is growing and so is your production, production rate, and is expected to be sustained, that's the point in time when you begin to say, "We know what a single dose will do. How about somewhat less but related doses, what happens, then? Does it affect liver function; does it affect the respiratory system," and so forth and so on, and those are usually done as an increment of what your market is like and what your projection for sustaining that market is.

If you're going to get into a product and it's going to disappear in two years, chances are there's probably not a whole lot of justification for that economically or otherwise.

If it's obvious your product is going to be around for the next 20, 30, and God knows how many years, and you're growing, then at some point you do it.

Beyond that, having done that, even assuming you've got relatively negative data, meaning, you know, subacute studies at levels which fairly high show absolutely nothing, I mean, come out zero as it were, which is kind of the rare occurrence, because usually any material has some sort of adverse impact. At some point, you're really asking what's the impact in terms of carcinogenisis, because, again, your population exposed is growing as is your yearly production, and you're making presumptions that it's going to be around for some sustained time period.

Well, what I'm saying is they reached the tier 3 level of a long-term study, as I understand production rates, somewhere in the '50's or perhaps, at the latest, the early '60's. That was the point in time when people should have got off their butts and said, "If you'll kick in 25, I'll kick in 25," and so forth, and go out and sponsor these and see, and if there is, we'll know, and if there isn't, we'll all feel better.

Q. And it's your testimony that these types of studies were not done; is that right?

A. Well, if they were, they're well hidden in the literature. Now, there's no studies of those types done.

The studies that were done on vinyl chloride are almost an academic curiosity because Viola was looking for the bone changes in the extremities, and he accidentally turned up the fact that at the highest concentrations, not only did he produce the bone changes, but he also had a few cancers.

That then gets pursued in more depth by Maltoni who confirms the bone changes, and then says, "By the way, I got my cancers at a much lower concentration because, because, because."

By the time the NCA study got out, the Maltoni data was practically available for all practical purposes, as I recall, and again, there was still that three-year gap in between the Viola work and even initiating the MCA studies for various and sundry reasons.

In the meantime, nothing gets done in terms of air monitoring, improvement of product qualities, and you get the distinct impression in reviewing the depositions that everybody's waiting for the other guy to take the initial step.

Q Are you done?

A I'm finished.

Q Where did you get your information regarding

what was being done by the NCA between the time of the Viola presentation and the time of the OSHA hearing?

A. Probably the best description is Wheeler, since he was head of the committee.

Q. Do you know Nick Wheeler?

A. I don't know him personally. I knew the co-chairman -- or I should say I know but I haven't seen him in years.

Q. Who is that?

A. That was Norm White, Shell Chemical.

Q. With regard to the long-term studies, can you tell me what long-term studies were being done by you at Sun when you were there?

A. Yeah, we had -- we had long-term carcinogenetic studies going back -- well, as an individual company, back as late as the late 1960's. However --

Q. Wait a minute. The late 1960's?

A. Yes.

Q. You're saying the '50's and '60's?

A. Yeah.

Q. What studies were being done in the 1950's and 1960's?

A. Okay, I'll clarify that for you. In the late --

well, let me clarify that. I think, believing if my memory serves me correctly, starting in about the mid '50's, Sun and API--because, again, they did that through the trade association, which is the logical way to do it because they're expensive studies--did a number of long-term bioassays on various oils and oil products at Kettering by Portman and Bingham, Eula Bingham I recall her name was, but they date back, I think, to maybe the '50's, late '50's.

Q And these are the experiments where they're painting the tars on the mice?

A This is the experiment they were doing skin painting and so forth and so on.

Q And they derive cancers from that?

A They derive some lovely cancers.

Q What about the materials that there was no indication from the people using them or otherwise that they caused any cancers? Did Sun Oil test those materials again?

A Yes. Oddly enough, we had in our late '60's studies anything from materials which we fully expected to be negative and weren't, to some materials which we knew would be extremely positive, and they were more

positive than we thought they would be, and those in between we really weren't sure.

Q. How long were those studies?

A. Eighteen months. That was classical in that time frame. It's only been since the early '70's that we've extended them out to two years and more.

Q. You were doing studies on benzene?

A. There were studies done on benzene. I think the studies on benzene go back even before that, but more importantly on benzene, we already knew in the 1920's that benzene was leukemogenic.

Q. From the occupational experience?

A. Yes.

Q. And you didn't have that advantage with the vinyl chloride workers, did you?

A. Well, the answer is yes and no. You got to remember benzene causes a specific type of leukemia. Had you specifically known from possible animals ahead of time that angiosarcoma was a likely outcome, then you probably could have looked at it and spotted it much sooner than it was in late 1973-1974.

Q. So what you are saying, with the benefit of hindsight, we can always know what's going to be --

A Well, if the animal studies took place, and assuming what Maltoni saw and Viola saw was seen in the late '50's or early '60's, you would have almost said "Wait a minute. We should be looking specifically for angiosarcoma as the best, most sensitive indicator of a potential problem."

The problem you have in, quote, "cancers" are if you don't carefully diagnose them, you may end up saying, "Well, the standard mortality ratio here is about normal to here," and if you look at Tabershaw's studies, which are perfectly good studies, there's no increased cancer incident in those people retrospectively. Well, somebody said how do you account for -- and we then think, "Well, now that you mention it, the angiosarcomas are a very rare form of cancer," and that is only reason it got picked up, fortuitiously.

Q Tell me, was there any evidence of cancer among the PVC manufacturing workers different than that which would have been expected before 1974?

A Well, the answer is not a hard yes and no, and it's not for this reason: If you do epidemiology studies on selected healthy male adult populations, assuming you have no cancer risk, assuming you do, and you can apply

that to other than cancer as well, your standard mortality ratio will almost consistently run far below unity, meaning they'll be 80 instead of 100 or some multiple on the hundred.

It's only where you have a traumatic increase in cancer, either a specific type or some other -- almost some other health problem that you'll see it simply because you're dealing with what we call a healthy worker syndrome; that is to say you don't hire the people, or you don't tend to hire people who are sensitive.

You don't tend to hire the people who are subject to medical problems, therefore, you would invariably, unless you have a problem, have standard mortality ratios less than a hundred, so if you see something 110, you have to admit that statistically that doesn't mean anything.

The other side of the coin is, well, that's only because your ability to measure it is limited, but if, in fact, you really expected 80 and you got 110, while present statistical models don't allow you to put significance on it, there's no question that's way out of line. You just don't have big enough numbers in which you can apply it to everybody's statistical satisfaction.

Think of the petroleum industry. We did total mortality-morbidity studies in the early '70's, and there

was no SMR's, with the exception of lymphoma, was consistently 75, 80, and guess what, the lymphomas came in 105, 110, 118, but you say that doesn't mean anything, but guess what, in animals, you can now reproduce the lymphomas, and, in some, address that. That's the exposure to some hydrocarbons.

Q I'm not sure that you've answered the question that I've asked you, so listen to my question, all right?

A All right, I'll try.

Q Is it true that in 19 -- before 1974, there was no recognition that there were any unusual rates of cancer arising among PVC manufacturing workers?

A Only because nobody had done any real studies to prove one way or the other, you know.

Q I understand your answer to be yes; is that correct?

A That's correct. But again, you're getting back to the old body counting phenomenon, if I can't count the bodies because I can't visually see them, there's not a problem then.

Q That's what I'm trying to explain, what the trigger signal is here. Isn't it also true, Mr. Todd, that polyvinyl chloride has been manufactured since the

1920's or 1930's with no unusual incidents of disease arising and being associated with that manufacturing process?

A Well, let me qualify that. The answer is yes but -- and the "but" comes in here.

We went into Air Products and initiated their occupational health program. Now, up until that point, their medical evaluation was really you bring a guy into the plant, the personnel man interviews him. He says send him to a G.P. who sees him and puts down the guy's in excellent health if that guy don't literally drop over or something or manifest some severe problems in terms of morbidity, they don't have a problem which indicates a damn thing, and, unfortunately, that's where a lot of the specific industry that you're referring to was up until January of '74, that really the medical programs were either next to nothing and the medical record retention and retrieval was even worse.

Q Okay.

A So, you know, how can you draw conclusions from somebody where you can't put the data out?

Q All I'm addressing, Mr. Todd, is the triggers which would have allowed the company to investigate this

particular material. Now, I believe you have agreed with me that they've used PVC for a long period of time.

A. Yes.

Q. With no obvious indicator that there was health risk involved other than the narcosis and explosion problem, correct?

A. Well -- I wholeheartedly agree from an acute standpoint, vinyl chloride appears very innocuous and that in terms of body count or some extreme morbidity, that did not show up. The same, by the way, could be said about lots of other materials which aren't exactly in our better graces today, including asbestos.

Asbestos wasn't a problem until back in late '50's and early '60's, at which time you had enough incubation and they began to blossom.

Q. And a lot of the products that we thought to be safe turned out not to safe, and we're going back revisiting the problem.

A. Well, again, if you look at asbestos, it's the same story of nobody doing the long-term studies and the medical evaluations until Sillicoff got his contract with the union and he finally published in the journal and he says, "Look what I've found."

Q. The ACGIH issued a threshold level of 500 parts per million for vinyl chloride up through the late '70's, middle '70's, anyway; is that right?

A. No, no. The 500, if my memory serves me correctly, was implemented sometime, I think, in the late '50's.

Q. Well, I'm asking when they changed it.

A. But it changed, as I recall, around '68 to '70 to 200, and I may be wrong a year one way or the other, but by the time '74 came along, I'm pretty sure that that 200 was already off the tentative list onto a final list, and, in fact, my impression from being in those plants in the early '74 era is nobody was paying a damn bit of attention to everybody.

Q. I may be mistaken on numbers, and I'm not going to argue about that, but the point I'm trying to make, even the members of the American Conference of Governmental Industrial Hygienists, which includes the governmental and industrial hygienists, were not issuing any threshold limit values which would indicate that the exposure was dangerous or that there was a cancer hazard?

A. Yes, but I also don't believe ACGIH had access to the Maltoni or Viola data as it came in.

The only participants that I know that were made aware of it was NIOSH, and then in '73 in regards to those two studies.

Now, it didn't come to their attention, recognizing they are a review committee, and in some sort of substantiation whereby one or the other follows, there is a tendency for them to sit on it, that 500-200, as I recall, was a ration based on bone changes in the extremities.

Q. And the Viola study was at 30 parts per million; is that right?

A. I think it was closer to 30,000.

Q. I'm sorry, 30,000, excuse me, yes. Do you know how close that is to the explosive hazard?

A. That is somewhere around 90 percent -- 80, 90 percent of the explosive limit.

Q. And it's my understanding of your testimony that that study should have triggered the PVC manufacturing companies to do their own investigations; is that right?

A. Minimally, at least begin to look at means of reduction, reducing the PVC, but more importantly, looking at, then, our in-house structure, where are they in regard to it, and I think the assumption was made with that

30,000 ppm, nobody has a TWA of 30,000, and, therefore, we can extrapolate down to where our 500,000, whatever they believed their exposure might be, and we have a huge safety factor.

Q Well, before they started reducing it, I believe your testimony was they would begin their own animal studies and epidemiologic studies; is that right?

A No. When I said "reduction," I -- I said two things: One, reduce it in the PVC, if, in fact -- or begin to develop processes to do that; and, two, begin to look inhouse and see where you are and what your options for reduction are.

Q Well, why should you start reducing it when the only evidence you have is that cancer started at 30,000 parts per million?

A Well, because, again, you're going along with those blinders on you saying vinyl chloride is all my people are exposed to, and what I'm telling you is there may be a few circumstances in this world where there's an occupational setting where somebody's re-exposed to one substance alone, but in the real world, it's usually a full host of materials, and I don't care how good your animal studies are, nobody is going to study all the combinations

and materials as they exist in the real world.

Therefore, to say willy-nilly,well, at 30,000 ppm, don't worry about that because that's rats or that's some rodent, and now I'm going to pretend I have a pure exposure, in their industries, since people are exposed to 1,000, there is a 30-fold factor difference, forgetting there are other air contaminants which will alter that, that is the fallacy.

Q So you're concerned with the synergistic problem?

A Well, potential synergistic or additive. You know, we don't know the answer to all these combinations of materials. If it were two per chemical homologue, maybe we can make some scientific extrapolations.

When you start getting materials which were totally foreign to each other, and in some cases on which you have very little data, you haven't the vaguest notion what is going to happen to your population.

Q After Viola, when should the companies have started doing what you talked about, and by saying, "You talked about it," I'm assuming that you're saying they should have started reducing their --

A Yeah, I'm saying the MCA committee that was

formed could have just as easily have said, "Let's go back and begin to develop processes for producing vinyl chloride monomer in the process; two, let's go back inhouse and begin to develop monitoring methods, and actually go out there and do it, not walk around with explosimeters which tells you it's over a thousand ppm or less than a thousand ppm, as though a thousand ppm was some magical safety valve, and that should have been done in '70, maybe '71 at the latest.

Q. Let me understand your position now. You're saying that it was not good science to attempt to confirm the Viola study?

A. No, I didn't say that it wasn't good science. What I did say was it was bad science to get around to it two and a half or three years later.

Q. So you find it acceptable that the companies attempted to confirm the Viola studies?

A. Oh, I think it's fine to confirm the Viola studies, but just don't make that the only place you put your money and time and effort into.

Q. Do you agree or disagree that they have attempted to confirm the studies before determining whether to act or not?

A. I agree they should have confirmed the studies. I disagree that they waited until they confirmed the study before they act for two reasons: One, assuming they could get the study started immediately, you got to wait two years for results; assuming you don't, you have a far longer time period, and that's exactly what happened. They ended up with something closer to three and a half or four year time period.

Q. And you are disagreeing with Dr. Viola, then, when he said that his animal studies should not be taken as evidence of action or equivalent action nor effect on humans?

A. If that's what he actually said, I disagree with him. I'm not sure that's what he actually said. That's what is stated in a number of depositions.

Q. Well, if he said that, you disagree with him. You're taking his data, but you are not taking his interpretation of the effect of that data?

A. Yeah, I disagree with that. Again, I only had one concentrate, and as I understand the data, not having looked at it, knowing he extends them to one concentration and says, "Don't worry about this. It is very high concentration, and there's no problem posed,"

and so forth and so on. That assumes that he understands what exposures are really like. I'm not so sure he knew enough about what the exposure circumstances are.

Q Why do you say that?

A Well, first of all, I don't know why he picked 30,000 and 30,000, period, if that's what he did. You know, typically, you want at least two or three doses to see if there's some sort of linear or nonlinear response.

Two, what he was initially asked to study, as I understand by the European manufacturers, was is there a relationship between the monomer and these skeletal deformities.

Now, his studies strongly suggest there was. However, either those manufacturers told him that they had people walking around in 30,000 ppm, or he wanted to be sure he didn't miss it, well, that's fine. If the latter was the case, you would still do multiple doses.

I can only assume one of two things: Either somebody was very thrifty and says, "We don't want to spend much money. Run one dose." Well, I'll run something that's somewhat less lethal and won't blow up on me, but will give me a response one way or the other; or, in fact, he's the sort of individual who didn't really believe the response

could occur, and, therefore, run into one dose, and I kind of doubt the latter.

I think what happened, they said, "We don't want to spend very much money. Run one dose to prove or disprove the relationship." But 30,000, admittedly, is so high that you wouldn't expect TWA's of 30,000. Even I've never seen that, although, by the way, I have seen short terms of 30,000, believe it or not.

Q. So as I understand your testimony, the Viola study was not as good from a technical point of view as you would have wished?

A. From a technical standpoint, if you were going to sponsor that study for a trade association in the United States at that point in time and you had some decent toxicologist providing input, they would have shot it down in a hurry and said, "Why one dose?"

Q. Do you know whether or not that was done?

A. I haven't seen direct data from the study or much less input on its origin. I'm not even sure that's available.

Q. Let me ask you something else: Do you take the results of a study that is derived from an interim report, or do you wait for the conclusion of the study?

A. It depends upon what the interim report says. If the interim report says there's not much of a problem, then, obviously, you can well afford to wait for the final results.

If the interim studies or records suggest there is a significant problem, then I don't think you can afford necessarily to wait. As a matter of fact, it's not unusual in toxicology areas to begin second studies during the course of an initial study, because some phenomenon is showing up that you didn't anticipate, and now you have to redo a second study to evaluate that aspect.

Q. Such as lower exposure level?

A. Such as lower exposure level or looking for a phenomenon clinically or otherwise that you hadn't considered in your original design.

Q. Have you talked with anybody in the industry about this period of time between 1970 and 1974?

A. Well, obviously, I talked to people in SPI who were members of SPI and who had products where we had actual field experience as well as talking to the staff.

Q. And what did you learn from those discussions?

A. Well, my impression from them in 1974 was, again -- and again, I don't think Air Products had a very

active membership in SPI, or at least paid too much attention to what was going on in the industry because they were kind of surprised by the angiosarcoma phenomenon, and, as a matter of fact, up until that point in time, hadn't done any monitoring, either, absolutely no monitoring.

Fortunately, the basic methods to do it predate 1974, so it didn't require a great deal of modifications or new equipment to be able to accomplish what needed to be done initially, and, of course, that's become more sophisticated with time.

Their basic process, like many of them, was wide open. You couldn't have put sloppy ventilation together if you intentionally wanted to go out and do it, and I think the premise was here's a material that is relatively innocuous, rather pleasant smelling; acutely it looks just a little more harmful than water, and if it's the case, there really shouldn't be a problem, and, therefore, we can afford to continue as we have in the past.

Q Other than the people at Air Products, did you talk to anybody about animal testing or epidemiologic studies which might have been suggested between the time of the Viola study in 1970 and the Goodrich report in 1974?

A No, I haven't specifically talked to anybody

who probably would have input to them, the possible exception being, and I don't see him any more these days, is Norm White.

Q. The only information you have is that that's been provided to you by Plaintiff's counsel?

A. Well, yes and no. Again, some of that I was aware of long before it was passed to me in terms of what's gone on in the PVC industry pre 1974.

Q. What was that again; what did you know?

A. Again, from my experience in talking to various SPI representatives, people who I know professionally, okay, as well as being in the Air Products plants themselves, what they describe in those depositions is pretty well consistent with my own experience in the field, so there's no big surprise on that.

MR. BUNDA: Let's stop for a second.

(Whereupon, Defendant's Exhibits 3 and 4 were marked.)

BY MR. BUNDA:

Q. Mr. Todd, do you have any information concerning any allegations of a conspiracy to suppress the information concerning the carcinogenic nature of vinyl chloride?

A. I don't know that I would call it "conspiracy," quite. That may be a matter of individual interpretation.

You know, if you read Wheeler's deposition and some of the information that goes along with it, apparently when the Viola situation came up, there was rather over-concern particularly in regard to the impact of the Delaney amendment, which is related to food additives and food contact uses, and their concern was that if the material, meaning vinyl chloride, could be demonstrated to be carcinogenic in animals, it could literally be banned from any direct food contact or food application uses, which represented a tremendous part of the market.

I'm not sure that you would call it a conspiracy, but depending upon how you want to interpret it, obviously, they had more than just a scientific concern there. Their concern was a loss in the marketplace of a good share of their product, and, therefore, there was a tendency to say, "Well, we're going to do some more studies, but let's don't rush out and do everything at once."

And over, of course, a two-year period or so, they finally go around to sponsoring the NCA studies and biotests.

Is that a conspiracy, I don't know. Some people, depending on where you sit on the spectrum of conservative to liberal might interpret this as conspiracy, other ones as bad judgment, procrastination, and something in between.

Q How would you characterize it?

A I would characterize it minimally as lousy scientific judgment. You know, you're covering yourself economically, but you're not covering your population in a realistic fashion nor in a timely one.

Q Apart from how you may judge the information that you've been presented with by Plaintiff's counsel on this issue of the lag time between Viola and Goodrich, do you have any information available to you that would be relevant to the issue of a conspiracy?

A Well, I don't know. Again, that may be a matter of interpretation, and I don't want to put things in a little compartmentalized box, but if I were in that situation, I would have done exactly what I referred to earlier in terms of doing things in a timely scientific fashion, but I think I would also have, at the same time, implemented that reduction I talked about. Even if you didn't inform the users of the products, at least you're

providing them with some much earlier protection than, in fact, they really did, so minimally that.

Now, if you really wanted to go in the other direction, you would go on, say to them, "Hey, guess what? I don't know what it means, but I have some indications this stuff may be carcinogenic, so treat it as though it might be, and if we're wrong, we'll come back in a couple years and tell you we're wrong, and we'll all feel better."

Q Listen to my question, Mr. Todd.

A Uh-huh.

Q My question is not your opinion about what was done or what was not done or what could have been done. Let's leave that to one side. My question to you was do you have any information which would be -- not opinion now, but information which would be relevant to this particular topic other than what you've been presented with by the attorney for the Plaintiff?

A I thought you were asking me earlier whether I would characterize that as criminal negligence.

Q No, sir. My question now is, and I thought I was asking it, and if I wasn't, I apologize, but my question to you now is do you have any information, any facts known to you other than that which has been contained in the

depositions or in the other materials presented to you by Plaintiff's counsel pertaining to the issue of an alleged conspiracy?

A. I think the answer is no. What I have got in terms of hard, written information is what is in the depositions, what I know by reference from context is not dissimilar.

Q. Thank you.

A. But it's not hard data or hard information in written form.

Q. Defendant's Exhibit 3, can you describe that for me, please?

A. Well, I would say right off the top of my head that it's the vinyl chloride TLV documentation. I would assume it is the latest documentation, but let me see it -- well, if it's not the latest documentation, at least it postdates 1979, so I would think it is the latest documentation.

Q. All right. Not to be misleading about this, this was presented as Exhibit 80 in the deposition that you gave in the Columbus case in December of 1987. Does that refresh your recollection?

A. You mean in regards to the Columbus cases,

I think so, and if that's correct, the answer is yes.

Q. Well, in regard to that -- to what this particular document purports to be.

A. Okay, go ahead.

Q. You're familiar with the document?

A. Yeah, I'm familiar with it. I have all the current documentation in the office.

Q. In your opinion, does this represent the chronology and the information which was developed in the public arena concerning vinyl chloride?

A. What are we specifically referring to in the chronology?

Q. Well, I'm speaking of the whole document as it describes the development of information concerning the health effects.

A. Well, it tends to be chronological at least in terms of specific health effects, and, again, it, for example, brings up the Viola study, or references the Viola study of 1971, as well as his presentation of approximately a year prior.

Obviously, this wasn't referenced in 1968. I could be incorrect, but I doubt like heck that his '70 or '71 publications or reference in that reduction that took

place in about the 1970 era other than the one I referred to earlier from 500 to 200.

Q. In your opinion, is this an accurate chronology of the public information available on the health effects of vinyl chloride?

A. It's a good summary. That's what it's meant to be. It's sort of a scientific justification for the numbers in the background in terms of both animal studies and human response that they're aware of,either published or otherwise.

Q. If you wanted to get a summary of the medical information available to you as an industrial hygienist concerning vinyl chloride, would you go to the documentation like this on vinyl chloride from the ACGIH?

A. You might or might not. It depends. The documentation varies in the depth and quantity of medical studies included, various one form or another. They basically give a summary of experience medically, but not necessarily all the nitty-gritty, and so depending upon how in depth you wanted to go, you might start here, and then go on to specific references or other data bases--

Q. Well --

A. -- but as a starting point, it would be a good

one.

Q. Specifically with regard to vinyl chloride, is it then your opinion that is a good starting point for the medical information available on vinyl chloride?

A. As a starting point, recognizing that it tends to be a summary document and has to be restricted in the length of it and so forth.

Q. Where would you go, then?

A. Well, depending upon how in depth you wanted to go, you would either go to specific documents that have been changed out on vinyl chloride -- you mentioned IARC, and I believe NIOSH has changed out a document or two.

You can also again go to your scientific literature or your computer abstracts, and then retrieve your specific detailed reprints from that as you wish, and you can embellish it to the extent you wish, depending upon what the task is you're doing.

Q. Can I show you Defendant's Exhibit No. 4, then, please, and have you go through that a second?

A. Okay, hold on a second. I might have to get my glasses out. Either I have a lousy copy or my eyes are going bad.

This is a November NCA conference.

Q Do you remember where?

A I have a feeling it's attachments to Wheeler's deposition.

Q This was an exhibit in your deposition in the Columbus case again.

A It could be, it could be.

Q Did you -- I may have also picked it up elsewhere, but I think I have a copy of this somewhere else as well.

Q Had you seen it before the Wheeler deposition?

A Oh, yeah, I probably did, because those cases go back before these got --

Q Do you remember when you first saw this?

A No, not right off the top of my head. It might have been a year ago or so, maybe two years ago.

Q Did you have -- did this come to you from Plaintiff's counsel, or did you have the original copy that you gave to Plaintiff's counsel?

A Some of this I have seen sometime earlier. I've forgotten where I ran into it. It may have been on some nonlegal cases.

Q All right. There are two memoranda attached to this, Mr. Todd. One is the November 23, 1971, memorandum

A. Hold on a second. Hold on a second.

Okay, I guess that's -- this is one from Union Carbide, July 1973.

Q. Yes, sir, that's the second one.

A. Okay. Well, where is the first one?

Q. The first one was the November 23, 1971.

A. I'm sorry, I thought you meant attached to this. There's two total.

Q. Yes, sir, and as part of the same exhibit.

A. Yes.

Q. That was how it was presented to you in the -- in your deposition in the Columbus case, and you've continued that here.

A. I'm sorry, would you restate that again?

Q. They were attached to the Columbus deposition that you gave --

A. Okay.

Q. -- and I've taken that same exhibit and given it to you here.

A. I'm sorry, I misunderstood your question.

Q. Do you remember when you first saw the 1971 documents?

A. I can tell you where I may have acquired those

is probably through API, because, again, I was active in API until '76, '77.

Q Do you remember who you would have gotten it from?

A As a guess, probably from Shell.

Q And the 1970 --

A But it may have come right through the committee, because I was one of the active committee members. Also, my partner in crime was formerly the head of their committee on, for lack of a better term, health and safety.

Q That's the American Petroleum Institute?

A American Petroleum Institute. Again, there's a lot of cross membership between API, NCA, and SPI.

Q In the 1973 memo, do you -- have you seen that before?

A Are you talking about the one regarding the NIOSH meeting?

Q Yes, sir.

A I've seen both of these. Again, obviously, if you picked it out of an earlier deposition, I must have seen it, but I think it's in our records going way back to the mid '70's.

Q Is Dr. White still alive?

A. I believe he is. I haven't seen Norm for -- do you know -- I believe he's still alive. Doggone, I hate to say I don't know for certain, but I haven't seen any unusual obituaries.

Norm's got to be 70, thereabouts. I don't know the answer. If he is deceased, I'm not aware of it.

I believe I can say without fear of contradiction that he is retired. Whether he does anything for a living or not, I really couldn't tell you.

Q. In the 1971 memo, there are stars and underlining. Do you know who made those markings?

A. No, I don't.

Q. Those are not yours?

A. They're not mine.

Q. And in the '73 memo, the same questions.

A. Well, again, I'd have to know -- this may very well have come through API. There's -- those may be old markings. It's not my writing.

Q. Let me understand your testimony. You've seen this before you began working with the Murray & Murray law firm on this case?

A. I think I've seen this as early as the mid '70's when I was a member of API. I'm not sure when these came

out of our files, or whether they came out of the legal files, but I was aware of some of those NCA meetings and goings on, and if I'm not mistaken, the reason I was aware of it and had it in our files was because we were still working with Air Products up through, oh, '76, perhaps, as late as '77, and we would have kept that or derived that material for our own purposes at that point.

Now, whether this came through the legal files or from some of those old files, I'm not sure, but I was aware of some of those meetings and so forth long before getting involved in these cases.

Q. Did you ever have any discussions with Dr. White about this?

A. I haven't seen Norm to talk to, good gosh, I don't know how many years. Last time I talked to him was in regard to something I was publishing, and I think he was a reviewer or something, so I haven't seen him in years.

Q. Well, my question was did you talk to him at any time? The first memo was done in late 1971 at a time when --

A. I wouldn't have seen him in '71 anyway.

Q. Let me finish my question. You were in API at the time?

A That's right.

Q And the '71 memo, in any event, indicates that Dr. White was going to be part of a committee.

A That's correct.

Q Did you discuss this with him at that time?

A No. Norm White wasn't actively in API at that time, and therefore, I wouldn't have had access to him. There was a gentleman by the name of Oneil Banks who worked with Norm, whom Jack and I both know well, and if there was any cross reference at that time, it would have come from Oneil, O-n-e-i-l.

Q I'm not specifically referring to your API now and the --

A I wasn't a member of MCI, so I wouldn't have had direct access to MCI.

Q Listen to my question, now. Dr. White was appointed to at least a subcommittee on the NCA with regard to vinyl chloride. Did you discuss in any meetings that you had with him anything on this work that he was doing for that committee?

A I wouldn't even have been aware in 1971 that he was doing it. Even if that information was available in API, when it came to API's attention was at the point

in time just before it got formally broke; in other words, what I'm saying is in professional circles, if you're close enough to it, you know before the official announcement came out that it was going to come out and that there was going to be a mad scramble.

However, anything which occurred between '70 and December of '73, by and large, I found out retrospectively from things such as that which later got passed through API, perhaps that and a few other things which I no longer have access to.

Q. Well, let's change gears now for a minute. You went through the Chrysler plant this morning; is that correct?

A. That's correct.

Q. That's the first contact you've had with that Chrysler plant between October of last year and today?

A. That is correct.

Q. Tell me about that plant. Why did you go through it -- or tell me about that visit. Why did you go through it again and what were your impressions?

A. Well, two reasons -- One, in reviewing the depositions, it became obvious that there was relatively limited information on whether the materials were utilized.

Two, it was pointed out somewhere along the line that Dendinger did, in fact, not spend, by any means, his whole career in the actual Ink Room, and as a matter of fact, even Wallace spent a good share of his time as a color matcher and so forth outside of the Ink Room, per se.

So I wanted to do two things, minimally: One confirm some information that I was looking for in terms of other materials in use and how; and two, look at those other two sites above and beyond the Ink Room which I already looked at.

Q. What was the other information that you wanted to confirm?

A. When they talked about him working in a -- I guess you'd call it coating line, I wanted to know was this one of many or one of one, and if so, why on the coating line and so forth, and we got questions partly answered, but not totally to our satisfaction at this point.

Q. What was the information you received?

A. Well, it was a single coating line. Okay. Basically you got a premixer, and you're pumping it directly over for coating on the fabric. I'm not sure, and again, they'll probably have to go back and check where on the line they actually worked, whether it was

one place or several.

Q. Of what significance is it that it's a single one versus several?

A. Well, because if it was several, and somebody says, "I've monitored this coating line operator," first of all, I don't know what a "coating line operator" means. Is that the guy at the front end or the back end of it or something in between; two, is it one of six coating lines, and if it's one of six, how representative is it--Fortunately, it's one and only one, so that settles that--where is it in relation to the raw material and what kind of raw material is it; are we using bag materials; are we using bulk; if you're using bulk, is it a relatively closed system, or is it bagged and they're doing the usual manual dumping and so forth prior to introducing it onto the fabric.

Q. I'm sorry, I forgot. What was the second reason that you went out there?

A. So I'd have some understanding of the color matching operation as well as the area in which he worked above and beyond the Ink Room. This is for Wallace.

Q. And what information did you get from today's visit that was different?

A. Different, no big surprises, again, as you pointed out earlier, the color matching encroach laboratory facility's a little more automated than it used to be. They do things in a more quantitative fashion, but nevertheless, the basic facility is still what it used to be, and it's in the middle of the processing area and quite apart from the Ink Room.

Two, as you probably are aware from deposition, at some point, he moved up to that job, but at some point, he also regressed backwards into the -- I guess for lack of a better term, striker job classification where he did spend a greater time in ink and related formulation rooms making up the solutions for evaluation for color matching and so forth. So I wanted some confirmation on that, and I think, basically I got that.

Q. Who did you talk to today?

A. Well, Mr. Ferguson, and, again, I have a very poor memory for names, at least two of their supervisory people.

Q. Was there --

A. I still need some questions answered, so if you need the names, we will provide them, I'm sure.

Q. What questions do you need answered?

A. Again, I want to find out a little more about some of the materials they used, and in addition to finding out where the monitors were done, because as I pointed out here as to data from early '74, when they did get it in the program, and then it comes down to what does it all mean. You say, "I took a TWA sample on somebody," but what the heck did he do and where was he in that job description, which, you said if the guy is a machine operator, was he at the head of the line or the bottom of the line or somewhere in between or whatever is the case as best they can reconstruct it.

Q. Let me ask you something now about the cause of cancer. In your opinion, does the dose or amount of exposure that a person suffers to a potential carcinogen have an impact on whether or not that person is suffering a risk from cancer? Do you understand my question?

A. Yeah, you're asking me if there is a dose-related response.

Q. Yes.

MR. DELLI BOVI: I'm going to object to any questions that call for opinions to a reasonable degree of medical certainty.

If you feel able to answer the question, go ahead.

A. You're asking me if I believe scientifically dose response, and the answer is yes, but it's a more gray area when you get to carcinogens than within more symptom toxicology parameters. It's very easy to show a dose response to liver injury or something of that type, and you can almost quantify it as a function of concentration.

It is quite clear scientifically that the same sort of parameters, although I'm not sure that it's linear, occurs with carcinogens. What is your problem is can you scientifically demonstrate with most, if not all your population, what a true no-effect level is, particularly where you don't have a pure exposure, and therein lies part of your problem, because the reality of the work situation is it's rare when you do.

Unfortunately, we tend to do our animal studies and related studies in a relatively pure system and then make an extrapolation, so, yes, I believe there's a dose response relationship. I don't believe you can project that on a one-on-one basis in an individual, but it's clear the higher the exposure, the greater the risk.

Q. In your opinion, is the OSHA permissible

exposure level for vinyl chloride, that level being one part per million, is that a safe level of exposure with regard to the risk of cancer, given the medical information we have today?

A. I'm going to answer in a fashion which you may consider evasive. Scientifically, if it is a noncomplicated exposure, I believe that you shouldn't have cancer risk at one ppm or less. Again, your problem still lies in the -- in the softness of your data is individual susceptibility, but more importantly, what is the influence of other factors, either personal or occupational, and therein may lie your gray area.

But scientifically, I would think that you ought to be able to get by with a healthy selected population of one ppm or less and not see any increase in cancer.

Q. Have you seen the epidemiologic study that was done at the Chrysler plant?

A. No, I haven't seen a specific epidemiology study done there. I've seen the SPI and NCA sponsored Tabershaw-Coopersmith studies, and it's been a while since I've seen those.

Q. Have you seen those in connection with this

case or previously?

A. No -- well, the answer is yes and no. API had a number of studies going on about the same era, and there's a tendency when you retain an organization of that type to say, "What experience have you had before. Let me look at those studies and so forth," so I saw some of those studies back even before they were published in the mid '70's or that general time frame, and this predates the refinery studies done by Tabershaw-Coopersmith because they interviewed them as possible candidates for that.

Q. What's your opinion of that organization?

A. Well, that's a good question. Tabershaw, of course, is retired, and Coopersmith no longer does epidemiology to my -- I don't know, mixed feelings on them. When you talk to them, when you get also at the study, they'll say, "Well, that is what the data presents, but on the other hand, you can interpret it this way," and I was never quite sure when I was at API exactly what I was getting from them, but they sure know -- certainly know how to go about it in terms of nuts and bolts.

Q. Do you have any training in epidemiology?

A. We had a staff epidemiologist at one time, but we've since lost that individual.

Q. Do you have any knowledge concerning any world experience in epidemiology?

A. Well, Ennerline is the nearest one that I have done business with and have faith with. He's really a biostatistician epidemiologist. I'm not a walking encyclopedia on those. I know a few others by name, but not because I've worked with them directly.

Q. Have you ever heard of the name of Richard Munson?

A. No, that name doesn't ring a bell.

Q. Leonard Cheasi?

A. Again, I'd have to say I'm not familiar with the name.

Q. Sir Richard Dall.

A. That name I've heard, but I don't know where I've heard it.

Q. Who do you recognize to be national authorities in occupational medicine?

A. Oh, I don't know that I could point to any one individual or organization as an authority on occupational medicine, particularly in light of the fact that a lot of your prior shining stars have either retired or went to the Great Beyond, and I'd have to say in all

candor, you could probably ask my partner, but I can't think of a really outstanding individual or group in the occupational medical area today that I could point to and say they represent the ultimate in the art.

At one time, a lot of your trade associations had really top-notch individuals, but most of them have either retired or died.

Q What about Jack Petersen in the field of industrial hygiene, what's his reputation?

A I was going to say I hope you weren't going to give him an M.D. degree the way everybody does me.

I would say I would give Jack an A only because I'd expect him to give me an A.

No, Jack's a good fellow, and he's been around a few weeks, and he's knowledgeable.

MR. BUNDA: Why don't we take a break for a minute. I'll see if I can review some of my notes and some of the things that I've got, and maybe we can wrap this up pretty soon.

(Whereupon, a discussion was held off the record.)

BY MR. BUNDA:

Q. Mr. Todd, I understand that you made some notes when you went out to visit the plant; is that right?

A. That's correct.

Q. I've handed you what appears to be original notes. Are those yours?

A. Well, this -- these appear to be separate from these, unless I've switched pens. I suspect these were taken at a different time, and not necessarily at the plant.

MR. BUNDA: Can we mark those as the next exhibit, and then I'll ask you to give me some idea about what they say.

(Whereupon, Defendant's Exhibits 5 and 6 were marked.)

Q. Mr. Todd, can you identify Defendant's Exhibits 5 and 6, please?

A. Hold on a second. Let me see which one's 5 and which one's 6. Five is some scratch notes. I suspect that this -- these may have been from a phone conversation with Kirk or others in regard to the case.

Q. And what is six?

A. Six is some field notes from the preliminary survey of October 6th, 1987, in which we went through the

three ink formulation rooms, and which I detail a few of the exhaust control pieces of equipment.

Q. Did you make any notes this morning while you toured the plant?

A. If I did, they were extremely cursory, and hold on a second, and I'll answer your question, because we didn't spend that much time there.

Q. All right.

A. If you wish a copy, it covers that much space (indicating).

Q. Well, what does it say?

A. Well, it gives batch sizes and temperatures, and that's basically it.

Q. What are the batch sizes and temperature?

A. It's 3,000 pound tank batches.

Q. This is in the Ink Room?

A. In the Ink Room.

Q. And the temperature?

A. They have the temperature elevated to approximately 180 as a result of the heat of solution, 180 degrees F.

Q. This comes from the mixing, the friction of the mixing?

A Well, it's the friction of the mixing and I suspect the heat of the solution.

Q And this is information you got from the Chrysler employees?

A This is from the Chrysler supervisory people, yes.

MR. BUNDA: Kirk, can we substitute copies for the originals of his notes?

MR. DELLI BOVI: Yes.

THE WITNESS: Do you have your own copies?

MR. BUNDA: Yes.

THE WITNESS: You want me to retain the originals?

MR. BUNDA: Yes.

BY MR. BUNDA:

Q Can you please look and confirm for the record that the copies are accurate reproductions of the originals?

A Well, let's look at the short one first. Yeah, they're not bad copies, and I don't see anything that's deleted in terms of not showing up in adequate detail on that one.

Q. You're talking about five now?

A. I'm talking about five, and I'm looking at six right now.

Yeah, they're pretty good copies, much better than I normally get myself.

Q. If I can direct your attention to No. 5, please --

A. Uh-huh.

Q. -- that's the one that is headed that reads "Chrysler VCM Cases."

A. Uh-huh.

Q. What's the next line mean? It's got some dates, April 1, 1976, and October 6, 1975. Do you remember what that means?

A. I am not sure, but I'll give you my guess. I'm guessing that this is a phone call in which they're indicating mandatory respirator use at some point either between '75 and '76, or starting in '76, and my impression is it's probably between the two dates.

Q. You have further down on that page the phrase "Dept no inks?" Do you recall your thinking at that time?

A. That probably means department numbers, by the

way. No, again, that's probably the same phone conversation in which they're indicating the Ink Department was the -- where the individual or individuals who have been there indicate potential exposure of vinyl chloride by Chrysler. That's the only way I can interpret it. That's what the question mark is there since I didn't know what was meant by an "Ink Department."

Q. And then again you seem to be also asking how much PVC they used in the Ink Room.

A. Uh-huh.

Q. To the left you have "PVC flour," is that a type of description for the type of PVC used?

A. By "PVC flour," I mean was it a very finely divided material, and did they use all bagged material or partly bulk, and the answer is they use all bagged materials here.

Some of the material was actually present when we were on the site last October, and it's a finely divided material. It's not a milled type of material or a granular, if you like that term a little better.

Q. Did you ever find out how much PVC they used in the Ink Room?

A. Not at this point. That still remains to

be clarified. I know they make 3,000 pound batches, but it still remains to be seen.

Q. Below that you have what appears to be "VC monomers," and then 2 in parentheses; is that right?

A. You're right about it being VC monomer, and I'm not sure what the 2 in parentheses means.

Q. Do you remember what you were thinking when you wrote that?

A. No, I don't.

Q. I'm sorry?

A. Go ahead.

Q. Then "Dumping platform B2 Ink Room" --

A. No, that's B-Z, breathing zone. Apparently there was data from somewhere in the Ink Room indicating a concentration of less than .25.

Q. Did you ever get any more information on that?

A. Well, again, as there's no data from some of the monitoring done, I'm quite sure this is probably a preliminary phone conversation where I tend to make scratch notes of this type.

Q. On Exhibit 6 in this, you seem to be jotting down notes about ventilation; is that right?

A. In part, that's correct. A good share of it's

related to ventilation.

Q Well, what is -- what are the notes that pertain to something other than ventilation?

A. Other than the spacial arrangements and size of rooms, ventilation covers most of the rest of it. Again, we had photographs to supplement these. That's why the notes aren't more detailed in terms of drawings and so forth.

Q. I notice that in the materials you've presented, there are a packet of photographs.

A. Yes, they're -- I saw them here a few minutes ago. I assume you've seen those. If I'm not mistaken, I presume Chrysler had an opportunity to see those. They were taken by --

MR. BUNDA: Kirk, is that the old set or the new set?

MR. DELLI BOVI: I'm taking a look at them.

THE WITNESS: These are the shots that were taken when I was there.

MR. DELLI BOVI: These are the set that were taken November 30, 1987.

THE WITNESS: Then they weren't

there when I was there.

MR. BAMMAN: You had October 7?

THE WITNESS: No, they have November 7, '87.

MR. DELLI BOVI: They may have been processed November 7, '87. I don't know.

THE WITNESS: They were taken --

MR. DELLI BOVI: In fact, I believe that's correct, because those were duplicates of the originals, so that November date probably refers to a processing date rather than a photograph-taken date.

BY MR. BUNDA:

Q Mr. Todd, did you direct that any of these photographs should be taken; in other words, did you --

A Some I did and some he took without any specific -- you want to go through them?

Q Well, I'd like to ask you if any of those photographs have any significance to you.

A Well, the answer is yes, and I don't know

whether you want to mark these or not. The first photograph, because it displays the new slot ventilation on these blend tanks for making it clear on the platform.

Q That's a mezzanine platform?

A This is the mezzanine or platform level of the middle Ink Room.

Q And what is the significance of those?

A Well, again, it shows the new modified slot ventilation on either side of the opening, which was installed in the mid 1970's.

Q Anything else?

A Well, other than this showing a lot of dust accumulating and so forth from moving bags in and out --

Q You're looking at the second photograph. It's just another view?

A This is nothing more than another view, slightly different angle. Again, I didn't direct individual ones being taken.

Q This is the one with the left two runs on the left-hand side of the walkway.

A This looks like it's taken from the opposite direction, I assume. That's the way I'd interpret the picture.

Q. The one that you just mentioned, that's --

A. That's dust accumulation on your diamond plating. That's typical of moving bags in and out, material getting spilled and to some extent becomes airborne, and settling out there and so forth.

Q. The next photograph?

A. The next photograph, which is the first one -- I'm going to put these down.

Q. This is a picture of some barrels?

A. Yes, this is a picture of some drums, and this has to be your pigment area. I'm trying to decide whether this is the -- this is not the same room. This is-- had to be Room 3. That is not the -- yeah, not the storage room, but the finished product area, and I presume these are finished drums of product, either that or old raw materials.

Q. When you say "products," are you talking about ink?

A. I'm talking about ink, yeah, finished product ink.

Q. Anything else about the ventilation --

A. No, there's no ventilation -- wait a minute. No, I guess that's just a scale, but I thought for a minute I was looking at the ventilation in the back of the scale,

but it's so filled with pigment that that's not the case.

Q. In the next --

A. Here's ventilation of some of your dust equipment, and, again, the upper left corner, some of your mix equipment.

Q. Anything else of significance?

A. I don't see any of the slot systems for your drums I have a feeling they're in some other photographs. This is, again, a picture from the opposite end looking right down here.

Q. You're looking at the next picture now?

A. I'm looking at the next picture.

Q. It shows --

A. Same area but from a different direction.

Q. Three different storage tanks on the left --

A. Yep, three elevated storage tanks --

Q. And in the middle, there are three ground --

A. Fiberboard drums, fiber drums full, and then on the left-hand side, all of your ink or pigment drums, and it's difficult to tell which. I would almost have to assume that they were pigment drums, only because I don't see any exhaust slots here for your typical formulation.

Q. Anything else of significance in that picture?

A. No, it's a repetition of the others from the opposite end. This is your bag storage area out in your main plant where your raw material, PVC and other components come from.

Q. You're looking at the next picture showing pallets of bags?

A. That's correct, pallets of bags, some covered with plastic, some not.

Q. There's an exhaust fan there, isn't there?

A. There is a typical wall-mounted exhaust, I would guess, maybe a three-foot diameter.

Q. Now, this is in a different area of the plant than the Ink Room, isn't it?

A. That is correct. This is in what you'd call more of your main storage area.

Q. Now, you are looking at the next photograph.

A. Now we're back up on -- I'm trying to place this part of the mezzanine.

Q. It's a picture of the mezzanine, again, and some air ducts?

A. Yeah, yeah. The problem I guess I have is this part of the mezzanine is filled with pigments and so forth. Oh, I see, here's the other mezzanine. Here's

the one down from it. I know where I am now.

Q. Have any significance to you --

A. Well, again, you're looking at the slot exhaust systems they put on these large tanks.

Q. I'm sorry, are you finished?

A. This is your fill point when you open those, either pump materials in or pour bags of raw material in, and then, of course, to collect the dust and vapors as well in a more direct fashion according to them, if I understand them correctly, and we still lack details. We had some sort of canopy, hood over these previously, canopy or other type hood.

Q. Again, to provide exhaust or ventilation?

A. Well, to provide sort of. It's not exactly the most sophisticated control.

Q. What's the next picture?

A. It's a picture of labels, and this, obviously, has to be a series of labels, strangely enough, off of one drum. The one's a timing label and the other is tetrahydrofuran, TEF, and the third one is MEK. Now, I don't know whether all three labels are on one can. I assume it's a mixed ink formulation with all these solvents in it. It's the only explanation that I can think of.

Q Then the next picture is a different view?

A And I think it's -- I don't know whether he insisted looking up the head -- on next thought, that is not a can. It's a big mix vessel, and that's why it's labeled as containing all three solvents.

Q Those are solvents used by Chrysler in the manufacture of its ink?

A Correct, those dilutants used for making the ink spread evenly and so forth.

Q Let me ask you something about those exposures: There were exposures above the threshold limit value for some of those solvents back in the plant in the mid '70's, wasn't there?

A Yeah, I recall MEK was the predominant material airborne. Again, you'd expect that based on its air vapor air pressure, but again, I'm saying that based on recollection without having the data in front of me. That was the 1975-76 time frame.

Q And the ventilation was put in at that time to take care of those exposures.

A The additional ventilation was, yes. There was ventilation actually put in a year or more before that, and that was that March of '74 engineering and exhaust and

supply air modification that we discussed earlier.

Q. All right, and what is the last picture?

A. I can only assume that this picture, in addition to being these drums of finished inks, is also looking at this exhaust system along the wall.

Q. Does that have any significance --

A. -- which would be one of the original dilution ventilation systems.

Q. Other than that, does that have any significance to you, that picture?

A. Not really because you can't identify what's in the drums. The pictures are taken from too far away to tell anything, so all it basically depicts is the typical bun-type drum with what appears to be ink or pigment formulations, and that local floor -- not the local, but the dilution floor exhaust system used for fire control. I thought that there were more than that.

MR. DELLI BOVI: Just answer his questions.

Q. Have you seen more pictures?

A. I thought there was more pictures taken.

MR. DELLI BOVI: Let's go off the record.

(Whereupon, a discussion was held off the record.)

Q. Have you seen additional photographs, Mr. Todd?

A. If my memory serves me -- the answer is yes, but if my memory serves me, there was a series of photographs taken, too, prior to Wallace and Dendinger being deposed, which goes back a while, of course, and I've seen those as well. That's why I was saying earlier I thought I saw more photographs than that, but I do recall a lot of those were earlier.

Q. From your recollection, was there anything in those additional photographs which you deem to be important in connection with your opinions in this case?

A. Only that they show in a little more vivid detail some of the slot exhaust on the drum filling operation and so forth that these don't show, so they're complimentary, and as I recall, we looked at those and then we made the site visit. The attempt was made to compliment rather than duplicate the originals.

Q. Mr. Todd, with regard to any exposure of vinyl chloride gas from the PVC that occurred prior to 1974 when the additional exhaust ventilation was put in

by Chrysler, do you have any opinions concerning whether that gas dispersed evenly or whether it either rose or settled?

A. Well, in theory, it would settle, but that's only in theory. In fact, unless you've got concentrations which were fairly astronomical, you would have--with any sort of reasonable air turbulence and occupancy air, you'd have relatively uniform distribution.

I'm saying that if you did a very quantitative study, that you couldn't see a pocket here and pocket there, and certainly if you're doing a specific event which generates the material, and therefore radiates out from there, that you won't see that sort of phenomenon.

What I'm saying is that barring those exceptional circumstances, you wouldn't expect to see 99 percent down near the floor, even though vinyl chloride is much heavier than air, 1 percent in the top 15-or-so-foot elevation.

Q. Well, conversely, you wouldn't expect to see a heavier concentration up on the mezzanine, either, would you?

A. Well, the answer is yes and no. If you were doing specific tasks which would generate it, then

the answer is yes. If you were talking about attic operations and there was no point source emission up there, then the answer is no, so I have to give you a yes and no answer.

In other words, what I'm saying is if you were doing bag dumping, you would expect that as you opened those bags and dumped them, depending on how good the whole exhaust ventilation was,that your exposure potential at that point would be far greater than if you were standing 20 foot away or 30 foot away. I'm not meaning to imply that an hour and a half after you got through dumping those and you closed the lid, if that's what you did, that if you then went up there that the concentrations were still considerably elevated above the rest of the surroundings.

It is not that kind of a dead air space by any stretch of the imagination, nor does vinyl chloride stratify to that degree barring you have extremely high concentrations, so there's kind of yes's and no's in there both.

Q Do you know what the odor threshold is for vinyl chloride?

A Well, roughly, depending upon who you believe,

an odor threshold is sort of a subjective thing. As near as I can tell, I think it's in the area of about a thousand. I've smelled it, by the way, if that's the next question.

Q. No, it's not. Would you have suspected someone to have smelled it in an area like this at Chrysler?

A. No. One, I'd never expect to smell it at a thousand ppm, barring some set of leak circumstances. Secondly, they use MEK, and it has a very low odor threshold and very pronounced odor, and if I were to assume the'76 MEK concentrations were correct and could be extrapolated backwards, I don't know how you could expect to smell anything but MEK.

Q. Let me ask you now a question about parts per million, the residual vinyl chloride monomer measured actually in the PVC grain or whatever--

A. Particles, whatever.

Q. -- is termed residual vinyl chloride monomer; is that right?

A. That's correct.

Q. That's on weight-weight basis that that's characterized as parts per million by weight?

A. That's correct.

Q. What did you mean by weight-weight basis?

A. Well, if you had a kilo of PVC, 2.2 pounds -- we can visualize that as being roughly a gallon container or something on the order of 92, you would literally have your parts per million, one milligram of vinyl chloride in there, okay.

Let's assume that really is a gallon. I know it's probably a little less than a gallon in actual volume. If we had one part per million equivalent volume of air -- what the hell would it be with one two hundredths of a milligram, there's one 150 to 200 fold difference.

What I'm saying is one part per million weight-weight for vinyl chloride is about 150 to 200 times what it would be in air. Are you with me?

Q. On a weight basis?

A. On milligram or weight basis. One part per million air is one very small fraction of one part per million in liquid-solid, solid.

Q. The OSHA permissible exposure level or the threshold limit value, for that matter, is on a volume basis, isn't it?

A. That's correct, so again, you're talking about much lower concentrations, because you're talking whole percentages or percentages of total saturation. A million

parts per million -- what's the molecular weight of vinyl chloride, a little over a hundred. It's on that order of 92, so it would be roughly a hundred million and 92 -- well, a million parts by a -- a hundred grams and a cubic foot of vinyl chloride would weigh --

Q. They're not directly comparable weights?

A. Only if you make the conversion. They're comparable, but they differ by a factor of somewhere between 150 and 200. I could sit down and calculate it very exactly out on that order of magnitude.

Q. Don't you also have to know how quickly the gas is given off by the PVC?

A. You don't have a situation where you have a parts per million here in the solid, then suddenly, instantaneously, it all flashes off, because it's trapped in something porous and may escape, you can't say I've got a ppm solid, and, therefore, I've got a ppm airborne or some fraction. I think that's what Carbide or others were trying to come to grips with.

If you set a limit of residual RVCM, could you, at some point in a realistic sense predict what you would exceed the OSHA standard or the action limit or whatever number they were targeting for. That's what those

mathmatical models and some of those field studies were oriented to, trying to make a prediction and, hopefully, go back to OSHA and say, "Gee, we have it low enough so that we can, with 99 percent confidence, state that exposures over the action limit couldn't occur." If, in fact, you could confirm that, you would, in theory, get away from some of your labeling requirements.

Q I understand your opinion. I'm changing gears now. I understand your opinions with regard to when animal testing should have been initiated by the PVC manufacturers and when residual vinyl chloride levels should have begun to be lowered. I would like to ask you now if and when there came an appreciation that there was a hazard exposure to vinyl chloride in the fabricating plants, which I believe your testimony has been there was a realization that the exposure was lower.

A Go back and restate that question. I'm not sure I understand it. Maybe you better break it down into a couple parts.

Q Let me break it down. If the exposure level in fabricating plants is recognized to be lower than it was in manufacturing plants --

A Generally speaking, I think that is correct.

I think that goes without saying.

Q. Given those exposure levels, when should the companies have begun to do epidemiologic studies and/or animal studies to determine where there was a risk of adverse health effects to the people working in the fabricating plants?

A. Well, you would expect, ideally, if the manufacturer recognized and initiated those studies in their own facilities, they, in turn, would recommend to the fabricator whether they were their own plant or somebody else's. Your real problem was that the PVC manufacturers didn't even appreciate there is a potential problem in their own plants, much less the guys down the road.

Q. So you're ignoring the factor of exposure -- or exposure levels then --

A. Well, it's not totally ignoring them. What I'm saying, I believe, in essence, they ignored it. It was one of these, "Well, here's this little piece of data," but because those were high concentrations, regardless of what it tells me, I'll go back to business as usual.

Now, I could understand or appreciate the situation if they said, well, let's now act on, for example, this Michigan study where they -- they based on this study

done on acro-osteolysis, and they said, "Well, 50 ppm looks as though it would be a good line." That would suggest that you better go on and find out where you are in relation to 50 ppm, and then get your own house in order, and then maybe pass it on and say, "Well, we think 50 ppm is acceptable from our standpoint, but know what our concern is, and then we'll keep you abreast as more data gets in hand."

What I see occurring from the depositions and other sources are, "Let's sit on it for a couple years. We'll do some studies, and those guys don't even need to know because we don't even know if we have a problem, much less having a problem," and I think it's only when it gets to the point that the new standard begins to impact on them when they go out and say, "By the way, fellows, got some good news and some bad news. The bad news is the studies are coming in and this is going to affect you. The good news is we don't think the exposure's going to be that awfully high, but here's how you are going to have to determine it."

Q When, in your opinion, to a scientific degree of certainty, should there have been a realization that there was a risk of health to the workers in the PVC

fabricating plants?

A. With the data, it's very easy to play Monday morning quarterback. However, again, you can always err on the side of caution as long as you don't panic anybody in the process of doing it, and having said that, I guess the statement I would make is the same time you begin to look at your PVC manufacturing, you recommend the same for them as well.

Q. And the basis for that is, again, the Viola study?

A. Well, the basis for that is not only that, but what you're saying is I'm going to do some leg work to see what I can do in terms of monomer reduction, but I don't even know how far I've got to go, because I don't know what the health exposures are in your plant other than the fact that they are in mine. There's no data other than my professional opinion, and Jack's, I'm sure, opinion, and experience would be the same, which would tell a guy who is the manufacturer, the president of X Company that that guy has to have an exposure which is one very small fraction of mine, and the reason I'm saying that is because they didn't even know what the heck their own expsoures were like.

You can't go out and monitor exposures with an explosimeter, and again, if you go back to all that time frame, that four, five year gap, nobody's generating any air monitoring data on their own plants, much less telling anybody else to do it on their's, and the reason they didn't want to recommend it is because they didn't want to do it on their own.

I would have done them simultaneously, recommended that for two reasons, because if I were in the position of the president of X Company and making decisions to drop the vinyl chloride level, I'd want to have some index of how much Goodrich was doing at that end, whether it was part of my company or Chrysler or some other user.

So if I told them I have a concern of possible health impacts, don't get alarmed, because the data's preliminary, but we're going to be taking some steps to reduce the vinyl chloride just in case, while we're doing it, which we don't expect to be able to do overnight, we'd like to do some monitoring.

We'll give you methodology, and by the way, the methodology was already available in a general sense in 1970. Gas chromatographs didn't pop out of the woodwork in 1974, and as a matter of fact, charcoal or similar

adsorption tubes -- that is AD, not AB -- they didn't pop out of the woodwork in '74, either, so the basic methods for even doing that were around prior to '74.

Nobody was doing that. Everybody was walking around with explosimeters and saying, "The explosimeter may respond to 700 to 800 ppm. If I don't see less than 700 ppm, it's not a problem. It's as though it's not there."

And again, ironically, it's below the odor threshold, so, again, you don't have a bit of data, subjective or otherwise, but I would have done the two of them simultaneously. It would have been judicious. It wouldn't have been extraordrinarily expensive, and it certainly would have been good product stewardship.

Q. You said it wouldn't have been extraordinarily expensive.

A. No, monitoring isn't all that heroically expensive.

Q. I'm sorry, what?

A. It is not heroically expensive.

Q. What is?

A. Monitoring, to determine what your expsoure of various risk groups are. If you go back to the various

1970 era, we used to crank out in the petroleum industries, hundreds of hydrocarbon volatile related samples a year, five, ten dollars a sample. Now they're up to, what, 25 or $50 a sample, but even so, we don't, on an individual plant basis, crank out that many, and if you do it and do it correctly, you don't need to.

Q. To do it, you need to know there is a hazard that exists, though, or may exist?

A. Well, to do it--one of the purposes in doing it is to find out where you are. Again, with some materials, if you got a material that has an odor threshold limit down here and a potential hazard level up here, you don't have to do a whole of air monitoring to determine where you are. I can walk into a MEK plant, and quite often, you can tell without taking an air sample that you don't have a problem.

But if I had a material with a TLV similar to MEK and an odor threshold of a thousand, that's a hundred versus a thousand, I couldn't tell you a damn thing by odor except if I smell it, you're way the hell over it, and if I don't smell it, I can't tell if you're nine times over it or two times over it or one hundred over it, and that's kind of where you are in vinyl chloride in that

'70 to '74 era.

Nobody knows, nobody seems to care; therefore, why bother the guy down the line, because you're really not interested yourself.

MR. BUNDA: I don't have any other questions.

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CROSS EXAMINATION

BY MR. MEYER:

Q. Mr. Todd, by 1976, Chrysler had made extensive improvements in the ventilation system, correct?

A. That's correct, they made some rather dramatic modifications.

Q. Okay. In the work you've done for Mr. Delli Bovi in preparing for this deposition, I take it you've had an opportunity to obviously review a number of records and some discovery responses, and in general, did you familiarize yourself -- gotten an idea of a total number of pounds of PVC resin that Chrysler uses in a given year, an idea of the volume?

A. I'm saying this from recollection, the last figure I recall seeing are something in the many millions of pounds for the year, but I can't quote you a hard number.

Q. If a particular company supplied a total of 370,000 pounds over the years 1976 to 1980, that would be relatively small?

A. I would think it represents a small fraction, and, again, I'd have to know the total consumption during that period, but my recollection is that would be a small fraction.

Q. Okay. Again, if a particular company did not supply any resin at all to Chrysler until 1976, and the total supply did not exceed 370,000 pounds through the years 1976 through '80, and the RVCM content of that resin did not exceed one part per million by weight, do you have an opinion whether that particular PVC resin had an effect upon the Plaintiffs in this case?

A. If I accept your numbers at face value, and again, I'm not saying you are incorrect, but if you accept them at face value as being a very small fraction of the total used post '76, and again assuming one -- less than one ppm actual residual monomer present, I would think they have a very, very small impact. I'm not even sure you could measure it except by our more sophisticated analytical methods these days.

Q. If, assuming again that PVC resin in question

is documented as having a RVCM of one part per million by rate, how does that compare with other resins? Is that pretty clean stuff?

A. Well, it's either very clean stuff, or it's one of the unique processes which produces a very low residual monomer, because I've seen numbers -- well, of course, if you go back far enough, you have in a couple thousand or more range up to even some reasonable ones, which far exceeds one, which certainly are far lower than they were a decade or so ago.

Q. What kind of process are you familiar with that typically yields those one parts per million or lower results?

A. I believe, and I'm saying this from recollection, I think it's a microsuspension process that gives you your lowest residual concentration. Again, their imposing above and beyond the pure process itself some specializations to it.

Q. All right. I'd like to go back on the question of parts per million by weight versus parts per million by volume thing, and if I can go over something that you were questioned on, excuse me and bear with me a little bit, but I'm kind of confused by it.

As I understand the B. F. Goodrich model that was discussed earlier, was the premise behind that to develop a model that if we have a known RVCM in parts per million by weight then we can then extrapolate or predict a RVCM content in parts per million by volume in the ambient air?

A. You said what you meant to say, but you put the wrong words in there.

Q. Okay.

A. The model was set up, and presumably confirmed with actual field data, because it looks like it is a pipe tail shotgun picture with scattered data points, suggesting that if you had a known residual level of monomer present in your polymer, and you had a given temperature, operating temperature, you could predict 99 or 95 percent confidence, I believe they use both intervals exactly -- I'm sorry, the maximum you could anticipate airborne, airborne meaning VCM and no residual VCM, if my memory serves me correctly, they were projecting that if you got down to 20 ppm or less with 95 percent or greater confidence, you would -- well, put another way, with 95 out of 100 samples you took under field conditions, they would predict that your actual exposure would be less

than a part per million if residuals were less than 20,000. They did a whole range, and I think their studies were up to about 500. I don't believe in those graphs they indicated anything higher than that.

Q. And you say the airborne numbers, is that parts per million by volume?

A. Yeah, airborne are always parts per million by volume.

Q. Which is what the OSHA threshold standard is based upon?

A. That's correct. All occupational limits for vapors are expressed in volumes.

Q. Would you feel comfortable, then, that if a resin is documented as having a VCM content parts per million by weight of one or less, that it would not exceed the OSHA threshold limits for airborne figures?

A. I guess, in general, the answer is yes as long as the analytical work is based on sound sampling and sound methods. Yeah, I would not expect a one-on-one relationship; that is to say, one ppm, even though there is a differential of about 150 to 200 fold between air and weight-weight. Nevertheless, you're degassing does not occur so instantaneously that you could expect concentrations

to ever exceed one.

No, realistically, I can't visualize it. Now, if you said I'm going to flash it off, I'm going to stick your head in closed containers while I flash it off, then you could do that at 150 to 200, but that's not the real world.

The real world it generates, it moves away from the point of origin, in which you have ventilation which would move it away, anyway, but more importantly, when you get to heated operations, nobody sticks their head in the reactor. At least we hope they don't.

MR. MEYER: I believe that's it. Thank you.

MR. BUNDA: Thank you, Mr. Todd.

MR. DELLI BOVI: We'll reserve signature.

(Whereupon, the deposition was concluded at 4:55 p.m.)

ALAN S. TODD

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STATE OF OHIO)
)SS.
COUNTY OF LUCAS)

I, Casey Gotthart, a Notary Public in and for the State of Ohio, duly commissioned and qualified, do hereby certify that the within-named Witness, ALAN S. TODD, was by me first duly sworn to tell the truth, the whole truth and nothing but the truth in the cause aforesaid; that the testimony then given by him was by me reduced to stenotype in the presence of said Witness, afterwards transcribed upon a typewriter; that the foregoing is a true and correct transcription of the testimony so given by him as aforesaid.

I do further certify that this deposition was taken at the time and place in the foregoing caption specified and was completed without adjournment.

I do further certify that I am not a relative, counsel or attorney of said party or otherwise interested in the event of this action.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my seal of office at Toledo, Ohio, on this 2nd day of May, 1988.

Casey Gotthart

CASEY GOTTHART
Notary Public
in and for the State of Ohio

My Commission expires
December 2, 1991.

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NOTES

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Q What you're saying is within each one of these categories you have to meet specific criteria to establish whether or not that's a good prospective study or a bad prospective study. You have to catch all the follow-up. You have to determine that the exposures, for example, are closely defined. Is that what you're talking about?

A Yes. And I'm not trying to, you know, say they're good or bad. Sometimes you can't get it. That's all I'm saying.

Q But those are variables that you have to look inside within each study?

A Yes.

Q But as a general matter your prospective study would be the one that you would like to, that you generally would rely on if you had a difference between that or you had to choose between that and a proportionate mortality study. Is that right?

A Yes.

Q And then between a proportionate mortality study and a case study, you would prefer to have the proportionate mortality study and you would rely on that more than the case study. Is that right?

A Right, yes.

Q Have you ever heard the term "background rate" before?

A Yes.

Q What is that?

A Well, that generally refers to the rate of a process disease, death that's occurring in the general community, a situation where you assume no exposures or equal exposures, something, some variable of that.

Q There are causes of disease, or specifically with regard to cancer, that we don't know about. They occur either spontaneously or they're common to the population. Is that right?

A Well, there's always a cause, but we don't know what it is, yes.

Q You try and distinguish those from the cancers that are caused by a particular chemical or exposures when you do an epidemiological study. Is that right?

A Yes.

Q Is that what we are referring to when we talk about a standard mortality rate?

A Yes.

Q And those things are taken from a nationwide

study sometimes?

A Some group, yes. They can either be nation, state, city, county.

Q With regard to cancer, those figures are available nationwide from, what is it, the American Cancer Society. Is that right?

A Yes. There are several reporting systems that will generate that data for you, yes.

Q Do you know what that information is for Erie County, Ohio?

A For Erie County, Ohio?

Q Yes, sir.

A No, I don't.

Q Can I talk to you for a second now about your knowledge of cancer in general. Can you give me some idea of the most prevalent cancers among males in the United States?

A Lung, probably the most common; is the most common.

Q A lot of that is ascribed to cigarette smoking. Is that right?

A Yes.

Q Ones below that, then, what is the next most common?

A I think colorectal, although prostate is about the same, I think.

Q Colorectal is a very common form of cancer in males. Is that right?

A Yes.

Q What about cancer in the salivary glands? Where does that fall?

A Pretty low.

Q In terms of frequency you're talking about now?

A Yes. It's low in frequency.

Q Would you tell me your understanding concerning what the medical community knows about the cause of colon cancer?

A Yes. There's some feeling that diet is involved, asbestos, some chemical associated other than asbestos.

Q What chemicals?

A Some of the polycylic aromatic hydrocarbons, some tars and pitches, acrylonitrile, some familial forms of cancer.

Q You're talking now about genetics, right?

A Genetics, yes, some association of cancer with a disease like ulcerative colitis. That's prob-

ably -- I've left out a few chemicals, but that's --

Q Are there cases arising that science just doesn't know what the cause of the colon cancer was?

A Yes.

Q How frequently does that happen?

A That's the majority.

Q What about sinus cancers?

A There are several causes. Cigarettes -- I'll probably talk about head and neck cancers in this group.

Q You're throwing them together with head and neck cancers?

A Yes. It's probably easiest. Cigarettes, alcohol, and then some association with cancers we've talked about formaldehyde. There is data about vinyl chloride and other irritants, wood dust, woodworkers, furniture workers.

Q Now, you're talking about in the mouth and in the head area. Is that right?

A Yes.

Q In general, as opposed to specifically to the salivary gland?

A Sinus, yes, or the salivary gland, that's right.

Q Specifically focusing on the salivary gland, do any of these, leaving aside vinyl chloride, but do any of these others, are they recognized specifically as attacking the salivary gland or the parotid gland? I'm using those terms equivalently.

A That's fine.

Q Is that your understanding?

A Yes. Well, probably cigarettes and alcohol. Some associations with other chemicals, but I don't think they're as strong.

Q Are there salivary gland cancers that arise that people don't know what the cause is?

A Yes.

Q What percentage or proportion of that?

A Those are the majority.

Q You're in occupational medicine. Do you have an idea about approximately what proportion of cancers are occupationally related?

A Someplace between ten and 20 percent, I would say.

Q Where did you get this information?

A Through a review of the published literature. You know, the low end, I think, is five percent, and I think Doll will talk about five per-

cent. The high end is about 20 percent, and it gets someplace in between there, although I think Doll is conservative.

Q You're talking about Doll and Peto's study?

A Yes.

Q That's the one they did for the National Cancer Institute?

A Yes.

Q Which one is the 20 percent?

A Well, several. I don't recall the authors for those that have suggested it.

Q That was the President's Advisory Board?

A That's one.

Q The one that Doll says is trash?

A I don't know that he said it was trash. If you say so, all right.

Q Well, you're not familiar with that, in other words. Is that right?

A I'm not familiar with Doll's comments on it, no.

Q You're not familiar with the study itself that says 20 percent, either, are you?

A Not that, no.

Q So you would agree with me that at least 80

percent of the cancers have or are thought to have origin other than arising from the workplace?

A Yes, at this point. We may be proved wrong in the future, but data today will tell us that, yes.

Q Can you describe for me your knowledge about cancer? What is cancer?

A Well, cancer has to do with altered genetics such that the cell no longer behaves normally and will spread, divide in a bizarre and unusual fashion, essentially disrupting normal physiology to the extent that life is not possible. It implies that the cancer will spread past the site of origin to a site some distance from that original site.

Q What is the word used for that?

A Metastatic disease; metastasis.

Q What causes the cell to do this?

A Well, probably several stages, several steps. We know a fair amount, although still the precise mechanisms aren't well worked out, but what likely happens is several different events change, a change in the DNA, the basic genetic code, such that the cell affected no longer either makes proteins, enzymes or goes about body growth repair normally, then followed by additional changes in other portions

of the genetic code, the DNA, so that the either control, regulation of that particular area, that particular gene, no longer occurs appropriately, allowing for additional and more rapid growth.

We're basically talking about likely several different steps involved with clones of cells growing at first not necessarily malignant but abnormal, becoming progressively more abnormal until they attain malignant status, that is, the ability to spread beyond the local source.

Q We're talking about the loss of a control mechanism in the cell for reproduction. Isn't that right?

A That certainly occurs, yes.

Q When you say there's a DNA alteration, do you know why that occurs in cancer?

A In some cases it's quite clear what happens. A chemical, because of its reactivity, makes what's called an adduct. It attaches to the DNA permanently in such a fashion that that cell and all the other cells that come after it don't behave properly or normally. In other instances, it's not clear. Some situations, pieces get added, deleted, but it's not clear, the process.

Q You're familiar with research on altered genes?

A Yes.

Q There are a lot of theories going around about carcinogenesis. Is that right?

A Yes.

Q And research is going on at this time?

A Yes.

Q But it's not exactly established how that occurs in all instances?

A No.

Q Do you know the phraseology or the terms "promoters" and "initiators"?

A Yes.

Q Describe for me your understanding of those.

A Well, that's basically what I was doing. An initiator is that chemical that will start the ball rolling, so to speak, has ability to attach to the DNA, change the DNA, form a DNA adduct in such a way that they have a permanent change.

The promoter has to do with a chemical or a substance that will make alterations at some other part that will enhance the growth, development of this initiated cell or cellae.

Q Do you know how vinyl chloride acts as a carcinogen?

A Probably in both of -- many of the initiators are also promoters. Either vinyl chloride through its metabolism forms this epoxide that's quite reactive, retrofilally attaching to DNA, changing it, forming adducts. This is fairly typical of many of the carcinogenic materials.

Q You say it forms an epoxide?

A Yes.

Q So what you're talking about is the vinyl chloride is metabolized. Is that right?

A Yes.

Q Vinyl chloride is not a carcinogen, but it's processed by the body to form a carcinogen. Is that correct?

A Yes. That may be a real shade of difference there, but that's right. It's probably a metabolite that causes the problem.

Q The metabolite has been found to be formed in the liver. Is that right?

A Well, anyplace where there's cytochromes. And the liver is not the only site. The liver is the primary site for metabolism, and most of the cyto-

chromes are there, but the process, the ability to metabolize materials, exists elsewhere in the body, too.

Q Have you heard the term "metabolic pathway"?

A Metabolic pathway, yes.

Q What is that?

A It speaks to what we've just been talking about, that is, the steps and the changes that occur in a specific substance, chemical, that either renders them active or inactive. Life makes many different things, proteins, enzymes, and there's a metabolic pathway that starts with building blocks and makes those. Or, with respect to synthesized chemicals, proteins in the body, or exogenous chemicals, outside the body, they get integrated through a pathway.

Q It refers to a mechanism through which an outside chemical comes into the body and arrives at a certain part of the body where it does harm.

A That also may be included, yes. It has more to do with pharmacodynamics. The pathway specifically has to do with what -- has to do with its degradation or anabolic process.

Q If I can back up for just a second, the fact

that, for example, sunlight is known to be a cause of cancer, skin cancer specifically, you know that to be true. Is that right?

A Yes.

Q The fact that sun causes cancer of the skin does not necessarily mean that sun can cause lung cancer. Is that right?

A That's right.

Q And that's because you have these metabolic pathways, and there's no explanation for how the sun could cause any changes in the lung, because the lung's inside the body. Is that right?

A Yes, although I'm not sure I understand the metabolic pathway, what you're driving at in terms of that. The sunlight does not reach the lungs. It's stopped by the skin. The energy is absorbed by the skin, so --

Q There's some pathway between what happens on the skin once it's exposed to the sunlight and the effects of the sunlight, the chemical changes that occur and the eventual development of cancer. That's what I'm getting at.

A All right. I wouldn't call that a metabolic pathway.

Q Well, there's some relationship between what's known to be a cause of cancer and the place where the cancer arises. Is that right?

A That's right. Energy is absorbed and the energy from sunlight, ultraviolet radiation, produces genetic DNA alterations in some of the skin cells, and that's what occurs.

Q You'd agree with me that the fact that something is known to be a cause or suspected to be a cause of cancer in one particular part of the body doesn't mean that that particular substance will cause cancer in any part of the body?

A No. You have to be able to demonstrate that that substance can get to -- I mean, that would be vital. The substance or the metabolic product or some such has to reach an area for that to be a primary site for cancer.

Q A more specific example, there is knowledge that vinyl chloride can cause angiosarcoma of the liver. Is that right?

A Yes.

Q That doesn't necessarily mean that vinyl chloride is known to cause skin cancer?

A That's right.

Q Let's talk now about the effect that dose has on the formation of cancer. What are your thoughts on that?

A That's a difficult area. From a statistical point you can talk about dose having an effect upon the prevalence, the amount of cancer that occurs in a population. The higher the dose, the more cases you're going to develop. But by the very nature of a carcinogenic material, it's very difficult to find a dose that doesn't cause some problem, i.e., potentially cancer. We know that the body has the ability to repair potentially carcinogenic events. In other words, a DNA adduct gets made that the body is able to cut out. But in terms of threshold dosage, for example, I don't think that we can talk about safe doses, safe exposures for materials that are carcinogenic.

Q Why do you say that?

A Because of what I've just talked about. In other words, if something is carcinogenic, it means by definition that it has the ability to alter DNA and, therefore, that ability can occur at almost any dose. Thus, you can't really talk about safe levels.

Q Why do you say that ability can occur at

almost any dose?

A Because all that it takes is one molecule at the right site to cause a problem. Now, statistically you can talk about the more molecules you have, the more likely you're going to cause that. That's absolutely true. But we know that one molecule can produce the problem.

Q Theoretically, you're talking about?

A That's right.

Q That is, as a practical matter it doesn't work that way. Is that right?

A Well, with many substances -- we can show that single asbestos fibers are sufficient to produce mesotheliomas, and that's probably the easiest example, because it's very difficult to give a person one molecule of a substance. But you can give them one fiber in a laboratory setting.

Q Well, all of us are exposed to sunlight and all of us don't have skin cancer.

A That's right.

Q Correct?

A That's right.

Q All of us are exposed to vinyl chloride, for that matter, and all of us don't have cancer of the

liver. Correct?

A You'd have to go some to prove how much we're all exposed to vinyl chloride.

Q Well, would you accept the theorem that all of us are at least exposed to a molecule of vinyl chloride?

A That may be so, yes.

Q So there are at least the defenses in the body to fight off exposures like this?

A That's right.

Q So it's not necessarily going to happen that you get cancer just because you're exposed to a molecule of a carcinogen?

A Right, yes. I didn't mean to imply by anything that I said that that was the case.

Q And, in fact, the U.S. Government -- again, we're getting into areas of policy -- but permits exposures to known carcinogens at levels which the scientific community, or at least the government, believes are acceptable.

A That's right. You know, cases at about one in a million have been ruled -- you know, exposures that cause, one in a million have been ruled to be safe, basically.

Q What do you know about the exposures that Mr. Wallace and Mr. Dendinger had in the factory? I'm talking now about materials other than vinyl chloride or polyvinyl chloride.

A There were some solvents of the ketone type, methyl ethyl ketone, MIBK, methyl isobutyl ketone, some tetrahydrofurans, I believe, some toluene, perhaps some trichloroethylene, some dyes. I don't know the specific names of those.

Q Now, did you analyze these exposures in your considerations of cause, potential causes, of their cancers?

A Yes, I've taken them into account.

Q What about MEK? Is this known to cause any cancers of the mouth and throat or the colon cancers?

A Not known to be.

Q Not known to you to be?

A That's right. And I don't think to the medical community.

Q What about the MIBK?

A Yes.

Q What about that?

A I don't think so for that, either.

Q For either type of cancer?

A That's right.
Q What about THF?
A Not known to be.
Q What about toluene?
A Toluene may be a promoter.
Q But in your opinion, it's not an initiator?
A I don't think it's an initiator, that's right.
Q What about the relationship between those? You can have initiation and never have it grow into a cancer. Isn't that true?
A That's part of the theory. I don't think the answer's in on that. But in the one sense I think the answer's no. It may be a very benign, slow-growing type of cancer. On the other hand, that may be, you know, the initiation may produce the benign growth, for example, although, to my point of view, to be an initiator of cancer really has to be something more than something benign, but depending upon which particular theory you're holding as to how you would answer whether a cancer can develop without a promoter. And, of course, many initiators are promoters themselves.
Q I'm not sure I got an answer to my question.

My question is if vinyl chloride, in your opinion, is an initiator and you get another chemical as a promoter, an initiator can change the DNA characteristic in the cell. Is that right?

A Yes.

Q That cell can die if it doesn't reproduce. Is that right?

A That's right.

Q And that's the job of promoters, is to promote the reproduction of those cells. Is that right?

A Well, all right. I mean, it's not quite that simple. If, in fact, the initiated cell dies, then no promoter is going to bring it back. Okay?

Q That's right.

A So the scenario would be that an initiator causes abnormalities that in theory ultimately would lead to a cancer. The promoter brings that cancer forward in time.

Q And if you don't have a promoter, then you don't have the cancer growing?

A Or maybe it's growing more slowly and isn't going to develop for some time, a longer time down the road.

Q Or it may never develop?
A Or possibly that individual could die from something else first, right.
Q So if you have toluene, which is a promoter, the toluene could have been promoting the cancers in these gentlemen?
A That's possible.
Q Have you sorted out in your own mind the relationship between these two?
A I think so. I'm interested in more information as to how much toluene and so on was involved.
Q Well, let me suggest that at times the toluene exposure was in excess of the threshold limit values. In your opinion, would the toluene have been a substantial contributing factor to the cancers that arose in one or both of these gentlemen?
A It depends upon how much, how often that was occurring.
Q Well, explain that for me. How much would be enough?
A Well, was this once that it was over the TLV? Was this every day, constantly, eight hours a day, five days a week, it was over the TLV? All of those things. I mean, the more of the exposure, the

more likely that it became important in terms of promotion.

Q Well, tell me how much exposure would be significant and enough for you to decide in your own mind that it was a substantial contributing cause to the cancers.

A I think that if there was constant exposure to a hundred parts per million, for example, of toluene, you know, eight hours a day, five days a week, through most of these particular men's employment, that would be an important role.

Q What if it was less than that in terms of the length of time? How about a couple of years' exposure?

A That also could be possibly important. It would depend upon which years, what else was going on at the time, and so on.

Q You don't have that information yet. Is that right?

A No.

Q That's information that you would need before you make a decision?

A Yes.

Q Have you been provided with information

concerning exposures in the factory?

A No.

Q Have you been provided with information concerning exposures to vinyl chloride?

A Not specific measurements. I have general knowledge of the types of exposures that would occur in this type of situation, but I have not seen specific numbers.

Q Tell me your general knowledge about types of exposures like this.

A Generally, conservative would be one to five parts per million generally with peak exposures probably occurring daily, peak exposures of 25 to 100 parts per million, although both of these individuals remarked that they could smell vinyl chloride, which would mean probably considerably higher exposures. You know, the ability to detect vinyl chloride, some say, can be done as low as 400 parts per million, but many think it's much higher than that.

Q In your experience, has anybody in a fabricating situation documented that they could smell vinyl chloride and it actually was vinyl chloride?

A This is the most descriptive detection that I've seen.

Q Have you expressed an opinion as to whether, in fact, they were smelling vinyl chloride as opposed to something else?

A All I have is their description of it, so I can't say.

Q What about if you were supplied with information that an industrial hygienist from the State of Ohio received that information, checked it out and concluded that they were smelling the ketones or something else? Would that have any bearing on your analysis?

A Yes.

MR. DELLI BOVI: I'm going to object. You can go ahead and answer.

Q Okay. What effect would that information have?

A If a hygienist had made an evaluation, that would certainly speak to the opinion at the time of the evaluation. These gentlemen are dead, so it's hard to question.

Q It's hard to ask them what they smelled?

A Yes. All we have is their description that they smelled this sweet type odor.

Q If you had your choice between the analytic

measurement done by the State and by Chrysler versus their opinion, which would you place more emphasis upon?

A It would depend upon the timing of that. I mean, if they're smelling this and saying "I'm smelling vinyl chloride" and someone is there measuring and says, "No, it's this," then it's much more on what's being measured. If it's a historical thing, "We used to smell this all the time; we haven't smelled it in a while," and measurements are done then and say, "Yes, you're right; you're not smelling vinyl chloride," then the historical observation would have more credence.

(Recess taken.)

Q Doctor, we were talking about exposure levels in the Sandusky Chrysler Plastic Products plant. You have not seen those figures. Is that right?

A That's right.

Q If, in fact, the exposure levels were below the level of .5 parts per million, would that have a bearing on your opinion?

MR. DELLI BOVI: Objection. You can answer.

A If someone can demonstrate, yes, that expo-

sures throughout the whole period were never above .5, yes, that way.

Q How would that affect your opinion?

A Well, that would mean there would be essentially little or no exposures to vinyl chloride.

Q And that would affect your opinion about whether these cancers were caused by vinyl chloride, then. Is that right?

A Yes.

Q You would have to change your opinion and conclude that they were not caused. Is that right?

A It would be much less likely, yes.

Q We weren't finished going through some of the other exposures. You thought there was some trichloroethylene there.

A It seems likely, yes.

Q What about the effect of that exposure? Would that have any effect on the cancers?

A Possibly. There's some evidence that trichloroethylene is carcinogenic.

Q If there was exposure to that chemical at the plant, in your opinion, would that have played a contributing role to the cancers in these gentlemen?

A It's possible.

Q What about the dyes? There were numerous dyes used in the ink room where both of these gentlemen had exposures. Have you analyzed any role that these dyes may have played in the cancers?

A I don't know which ones they were. Dyes, you know, bladder cancer's associated with some dyes for sure. So it would be valuable to get information about that, about those dyes.

Q Don't you need that information before you can form a conclusion as to the cause of the cancers?

A Well, except that the vinyl chloride is fairly striking here and that sticks out in terms of causality.

Q But you haven't ruled out any other possible -- these particular possible causes?

A For completeness I should get the information about the dyes, yes.

Q What about their exposures to other substances and materials in jobs other than at Chrysler? Have you analyzed that?

A Yes, I have some job descriptions of other employment.

Q Have you found any exposures in those jobs that are of significance to you?

A I don't think so. No, I don't think so.

Q What about their lifestyles? Anything other than their occupations? Have you analyzed that for both of these gentlemen?

A Yes.

Q Have you found anything there that, in your opinion, would be a factor for contributing to the cancers?

A I don't think so. Neither one of them. Mr. Wallace was not a smoker. Mr. Dendinger smoked a pipe, claimed not to inhale it. So both have very little smoking history. Alcohol is not a problem. Diet looked like there's nothing special there, either. So I don't think there's anything socially that's involved.

Q Well, let's stop for a second, if we could. The discussion of diet is in particular connection with the colon cancer. Is that right?

A Yes.

Q Isn't it also a fact that there is not a full knowledge by the medical community about what particular factor in diet causes the cancer?

A That's right.

Q So the fact that Mr. Dendinger ate like the

rest of us or, if I can back up on that, the fact that Mr. Dendinger had a diet and it's not entirely clear what that diet is, how can you rule that out as a cause of the colon cancer?

A It basically means that the risk of diet is probably awash with the background; in other words, that there's nothing that sticks out. Fibers seemed fairly normal, nonexcessive fat, you know, meat diet. So there's nothing that sticks out in it, and you're left with, well, there's some hint that diet may be involved, but given other exposures that we do know about, my opinion would then be more for the vinyl chloride.

Q Well, let's get into the meat of it here. I am correct, am I not, that in both the colon cancer involved in this case and the parotid gland cancer involved with Mr. Wallace, neither one of those cancers had any particular characteristic which is known by the medical community to be associated with vinyl chloride caused cancer?

Do you understand the question?

A By cell type you're referring to it?

Q Yes, sir. Or by any other characteristic.

A Well, there's several pieces of information

that would, in fact, link their cancers to vinyl chloride.

Q Tell me about that.

A With respect to the salivary parotid, we know that the zymbo gland cancers in the laboratory studies were occurring as a result of vinyl chloride. The analogy from the zymbo gland to the parotid salivary is, of course, very obvious. Humans don't have zymbo glands, but the location and the function is quite similar. I have had a previous patient with oral cancer as a result of vinyl chloride, and there are some interesting case reports showing that.

With respect to colon cancer, some of the epidemiologic studies will show increased rates of colorectal cancer. The epi. study of the Chrysler facility itself will show significant colorectal cancer in individuals with possible exposure to vinyl chloride, and I think that lends considerable weight.

Q All right. You misunderstood my question.

A Oh, all right.

Q Looking at the cancer cells and where they arose in Mr. Wallace and Mr. Dendinger, is there anything characteristic about those cancers which would distinguish them from a person who had cancers

in the same site but who did not have exposure to vinyl chloride?

A You're right, I did misunderstand, and there isn't anything.

Q So if we look at Mr. Dendinger and his colon cancer, his colon cancer would look the same as someone else who had colon cancer but had no vinyl chloride exposure?

A Well, right, but --

Q Well, I want to establish that so it's very clear on the record. There's nothing you can look at in these cancers per se that you can say just by looking at that, "I can tell what the cause was."

A That's right.

Q I'm trying to distinguish that from, for example, the DES cases where, as you know, you can look at those cancers and they're a very unusual type of cancer, and if you look at that and see the cancer cell type, you know what the cause is.

A There are a few situations, but --

Q These are not among them?

A Right. Most cancers you cannot tell from looking at them. Most cancer looks like any other cancer at that site.

Q So for the completeness of the record, the parotid gland cancer is of the same type or the same characteristic that you can't look at the parotid gland cancer and say, "I know it's mucoepidermoid cancer," and knowing that, say immediately, "Yes, I know that's vinyl chloride"? That's not possible?

A That's correct.

Q What about Mr. Dendinger and his digestive system problems? I'm still on the topic now, you understand, of other risk factors involved with cancers besides vinyl chloride. Have you analyzed his digestive problems?

A Meaning?

Q Are you aware of them.

A Other than that caused from his cancer?

Q Yes, sir.

A Like his peptic ulcer disease he had in the past?

Q Among other things, yes.

A Okay. That is all that I know about with respect to previous gastrointestinal tract problems other than that directly related to his cancer, and I don't think that that has any particular bearing on the development of his cancer.

Q How much have you reviewed of the medical records of both of these gentlemen?

A Records on Dendinger from, I guess the first records are from Sandusky Memorial Hospital, April '84 through the last record is, I guess, at Fire-lands' Community, the 31st of March of '86.

Q As you sit here today, you're not aware of his kidney or gallstone problems?

A The kidney, but that's not gastrointestinal. I was not aware that he had any gallstone problems.

Q What about his diverticulosis? I'm not sure if it's -losis or diverticulitis, one of the two.

A I am not aware.

Q Would that have any bearing on your opinion?

A I don't think either one of them would. If those were, in fact, true, they wouldn't have any bearing on his cancer.

Q Obviously he was having problems with his digestive system, though. Would you agree with that?

A Oh, quite clearly. I mean, that's what lead to the diagnosis, I guess, ultimately, April of 1984.

Q Do you have any idea as to whether or not a patient who presents symptoms such as these involving various problems in his digestive system presents an

increased risk of colon cancer?

A No. These problems described in him do not. There are gastrointestinal problems that do, such as ulcerative colitis or particularly familial polyposis, but these particular problems are not.

Q Do you know whether he has a family history of colon cancer?

A None that I know of.

Q You say you don't know of any?

A That's right. I'm not aware of any.

Q What about Mr. Dendinger's exposure through his painting and things at home? Does that have any bearing on you?

A Well, the painting can be hazardous. I did take that into account. The problems from painting, are primarily neurologic, and I don't think have to do with his, either; in this case, his colon cancer.

Q Did you take into account his exposure to carbon tetrachloride?

MR. DELLI BOVI: Objection. You can go ahead and answer.

A Yes. I'm not aware that he was having a carbon tetrachloride exposure.

Q Assuming that he had cancer exposure, would

that affect or change your opinion?

A I don't think. Carbon tetrachloride, of course, causes problems in the liver, including liver cancer. I'm not aware of relationship to colon.

Q Have you taken into consideration any other environmental factors in his colon cancer?

A I believe so, yes.

Q Are you familiar with the literature which suggests a relationship between colon cancer and coffee consumption?

A I've seen reports. I don't think there's any strong association there.

Q It's your opinion that there's a strong association between colon cancer and vinyl chloride?

A I think it's stronger than coffee.

Q And for that reason you're rejecting coffee and accepting vinyl chloride?

A Yes. Nothing that I have seen really shows anything between coffee and colon cancer. There have been quite a few studies to negate that.

Q If there are affirmative studies, would you accept that as being a risk factor for him?

A For coffee?

Q Yes.

A I think those have shown to not be good studies.

Q So the studies that negate that are more persuasive to you. Is that right?

A Yes.

Q What do you do when there are conflicting studies like that?

A Well, you need to look at the studies themselves and what was the methodology. How did they document exposure, you know, ingestion of coffee, for example. How did they verify disease.

Q If there are conflicting studies, can you establish to a reasonable degree of medical certainty whether or not a particular factor is a causative element?

A You can't always, without doing more studies. Depending upon the strengths or weaknesses of the study, you frequently can come to an opinion.

Q Are you familiar with the conflicting studies involving vinyl chloride in colon cancer?

A Yes, I think I am.

Q You're aware of the fact that there are epidemiologic studies showing no increase in colon cancer?

A That's right.
Q And in fact, a lower than expected level of cancer?
A I've seen those reported, too.
Q How do you reconcile those with the studies that you're referring to?
A Well, I think the particular piece that makes this one so helpful is the study in this particular plant. I must say that I haven't looked exhaustively at all of the negative studies to be able to truly critique them, but I think this study is particularly strong.
Q If I can stay on an abstract level for a second, you've been asked many times during the course of the deposition that you've been giving to render an opinion to a reasonable degree of medical certainty. Is that right?
A Yes.
Q What is your understanding of what that means?
A That it's more likely than not. Not a hundred percent, but more than 50-50.
Q 51-49 would be --
A Might be.

Q In terms of what's available in the literature, what would you need before you would conclude that there's a reasonable degree of medical certainty that something is a causative element?

A Evidence for general carcinogenicity. In other words, you probably would want to be able to show mutations in bacterial systems, although benzene sticks out as an example that doesn't do that. Laboratory evidence, and then human evidence.

Now, you don't always have all of those. If you have bacterial and laboratory and case reports, that may be sufficient. If you have good epi. studies like benzene, for example, without laboratory or bacterial systems, that becomes sufficient.

Q Have you formed an opinion in this case that vinyl chloride is a cause of the cancer in both of these gentlemen to a reasonable degree of medical certainty?

A Yes.

Q What is your opinion?

A Oh, that it has.

Q Why?

A Some of this I answered earlier when I misunderstood your question. That is, with the sali-

vary parotid, the obvious ability for the vinyl chloride to contact this portion of the body.

Q Well, wait. Okay. Go ahead. I'm sorry. Pardon me for interrupting.

A That's all right.

The zymbo gland tumors in laboratory animals, the case reports of oral cancer in individuals with vinyl chloride exposure, the lack of the two primary causes of oral cancer, that is, alcohol and cigarettes, and the rather unusual nature of a salivary parotid tumor make, I think, vinyl chloride a pretty strong case for Mr. Wallace.

With the colon cancer, frankly, I don't think the evidence is as strong. He doesn't have anything striking in terms of social or nonoccupational causes. With respect to the vinyl chloride, there's certainly a mechanism for the vinyl chloride to get to the colon. Inhalation, expectoration, ingestion and/or a pass through the liver and extrusion through the biliary system all demonstrate the ability for it to get there in the first place. The epi. study from the particular site showing increased colorectal cancer.

Q Anything else?

A No.

Q What is your understanding concerning the length of exposure for both gentlemen?

A Mr. Dendinger started working at Chrysler facility in 1968, and his exposure to vinyl chloride in the ink room occurred off and on throughout his employment there until, I guess it was 1980. He estimated about 30 percent of his time was spent in the ink room where, I think, some of the higher exposures to vinyl chloride occurred.

Q Why do you say that?

A The description of the process, dumping of bags of the PVC resin into the vats and the general processing seemed to me to make that the highest exposure to any of the VCM, the residual vinyl chloride monomer.

Q Why do you say that?

A Dust was highest, concentration of resins in the air was highest, and that would seem to me that that would be the highest level, the highest places.

The other time was spent, the remaining 70 percent, out on production where there were also some exposures but probably not as great.

Q That's Mr. Dendinger?

A That's right. And Mr. Wallace was involved in production from '72 to '74, and then after '74 was in the ink room almost entirely until he stopped working. I'm sorry. Until '78. Yes.

Q So at maximum that's a six-year period. Is that right?

A For him, yes.

Q In your experience, is that sufficient to cause any type of vinyl chloride related cancer?

A Yes.

Q Where do you get your information for that?

A The review even of the study of that plant talked about cancers in individuals less than five years.

Q Okay. There we're talking about lung cancers and different other cancers, right?

A That's right. Those are lung and colo-rectal.

Q There are multiple exposures or causes for those, right?

A There certainly were more than vinyl chloride, that's right.

Q Chief among the lung cancer the cause is cigarette smoking. Is that right?

A Could be.
Q You don't know the causes of the cancers in those cases, right?
A What do you mean?
Q From the plant study you don't know the cause of the cancer in those cases?
A All you can show is it was production that would show the statistical relationships.
Q Between what?
A Lung cancers and those in production areas had higher rates of lung cancer than those not in production.
Q Well, let's look at the study and see what you're talking about. I don't have mine with me, so I'm going to have to look over your shoulder.
A Coating/finishing, statistical relationship. Certainly elevation overall. They don't give P values there for me to know exactly what the relationship was. Again, review of all cancers, certainly lung is up. You can't make too much out of that because there's no comparison. But I think that's certainly something else.
Q That's what you're referring to when you're referring to Chrysler showing -- you're on page --

A Table 2.

Q -- 12 of the record. Table 2 refers to 8 of Chrysler and U.S. is 4.67?

A Right. And specifically this of coaters and finishers of 4 versus 1.1.

Q You view that to be significant to you. Is that right?

A Yes.

Q Do you know what the cigarette smoking habits of those people are?

A No. Well, we don't know that.

Q Do you know what the -- okay. I'm sorry to interrupt you.

A In SMR data without knowledge of cigarette smoking and you compare to the general population, you make an assumption that the cigarette usage is about the same. We know that's fairly reliable, not as exact as you'd want it, but without data that's the best you can do.

Q But my question remains, you don't know the cigarette smoking habits of those four people, do you?

A Nor do we know it in the other group, you're right.

Q But it's a lot easier to find it in four people in the factory than it is in the whole population of the United States, isn't it?

A That's right.

Q And you can certainly, by finding that out, find out whether your assumptions are correct or not?

A Well, but then you're --

MR. DELLI BOVI: Well, let me state for the record that we have requested that data from the defendants, and the defendants have refused, at least to date, to provide us with it.

MR. BUNDA: Well, I think that's incorrect, Kirk. You never asked us for the cigarette smoking history.

MR. DELLI BOVI: I asked for a copy of the report.

MR. BUNDA: You've got a copy of the report.

MR. DELLI BOVI: The report included a computer listing of the cohort. That would allow us some ability to determine who these people are that are identified in these documents. You should have that, hopefully, when we get to Milwaukee.

MR. BUNDA: I understand that, but you've

never asked us for the cigarette smoking habits of these people.

Q You need to know that, Doctor, don't you, before you can determine whether their lung cancers are most probably due to cigarette smoking or something else?

A Well, you need to know more than just their smoking habits. You need to know the smoking habits of the total population at the facility as well as the population in the U.S. in general, because right now we are comparing apples to apples. We're comparing groups with presumed similar smoking histories to groups with similar smoking histories.

Q You don't know the exposure levels of the people in the coating/finishing department, either, do you?

A That's right.

Q I forget what we were talking about. Oh, the period of exposure.

You're relying on this study to tell you that six years is sufficient exposure to vinyl chloride to cause cancer associated with vinyl chloride. Is that right, Doctor?

A Yes. And if you look at many of the epi.

studies, you've got a five-year period of exposure, frequently sufficient enough to produce problems with cancers developing with an appropriate latency period down the road.

Q Which one are you talking about?

A Well, I don't them filed right here to --

Q Well, I would like you to show me the study that says that there are cases of cancer arising five years after exposure.

MR. DELLI BOVI: That wasn't the question.

A Yes. Five years of exposure.

Q After five years of exposure.

A Right.

Q And that is deemed to be a sufficient exposure to cause vinyl chloride associated cancer. Can you do that?

A I'm not sure that I can do it right now.

Q Well, can you send me a supplementary letter indicating that?

A Yes.

Q Or you can give it to your counsel and he can give it to me.

A It usually helps if you send me a reminder to do that. Okay?

Q That's fine. But as you sit here today, you can't name a particular study that you're referring to. Is that right?

A That's right.

Q Would you agree with me that a five-year or a six-year period of exposure is not the mean or average latency period among vinyl chloride associated cancers?

A Oh, I wasn't talking about latency. I'm talking about exposure.

Q Well, exposure periods, excuse me.

A I think that five years has been shown to be sufficient.

Q No. My question was not sufficient. My question was the average exposure period is much higher?

A Oh, that's right. You're right about that.

Q Can you show me an exposure period of five years and a level of exposure similar to Mr. Wallace?

A I think that's in the literature. I don't have it pulled right out to do that. But if you want, I will look that up.

Q Where in the literature.

A Okay.

Q Would you agree with me that the epidemiologic literature which exists indicates that a level of five years of exposure total and a level of exposure of one part per million or less does not indicate any risk of cancer for vinyl chloride?

A Yes. If the exposures have been less than a part per million, that's pretty minimal risk.

Q Now, are you of the knowledge that Mr. Dendinger was a color matcher?

A Yes.

Q When you say that he had exposures in production, are you aware of the fact that he actually had an office separate from production?

A Yes, although the vast majority of his time was out there on the floor matching colors, going back to the ink room, back and forth.

Q How do you know that?

A From his description of what he did.

Q You haven't seen the plant, have you?

A No, I haven't.

Q Do you have any information how residual vinyl chloride is given off from the resin?

A Yes, a couple manners. In the processing of the resin in the first place, if the vinyl chloride

isn't appropriately vacuumed out, basically, there can be a lot of VCM that lingers on the resin and is present throughout the processing. Each particulate, each dust particle, if you will, may contain and does contain vinyl chloride monomer inside of it, and that's quite difficult to remove during the processing of the PVC and care needs to be taken then in its use so that any monomer that's generated as it's being used is also eliminated through engineering techniques, hygiene controls, or what have you.

Q Do you have any knowledge about the difference between the types of resin and the level of monomer they may contain?

A No.

Q Do you have any knowledge about the types of resins used by these two particular gentlemen?

A No, I don't.

Q With regard to Mr. Wallace now, and I'd like to restrict my questions to him for the time being, you mentioned as the first factor, in your analysis of why vinyl chloride was the cause of his cancer, the fact that there is an ability of vinyl chloride to contact this particular area. Do you recall that?

A Yes.

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Q What do you mean?
A Through inhalation vapors are in contact in the oral cavity and have the ability to be in contact with these particular glands.
Q How does that happen?
A Because of their ducts into the mouth themselves the monomer possesses the ability to enter them.
Q How do you know that?
A Well, I guess I have not specifically studied or seen someone trace vinyl chloride in that fashion, but we know that other materials that cause oral cancers, exposures are basically the same and that that's a presumed mechanism for contact.
Q Well, you're talking about, I presume, cigarette smoking and the cause of oral cancer.
A That would be one, yes.
Q And the fact that the cigarette smoking is in immediate contact with the tissues in the mouth?
A Yes, or with the tissues immediately adjacent to the mouth through ducts, and so on.
Q Haven't you already told me there's no specific association between cigarette smoking and parotid gland cancer?

A I don't think so.
Q Okay. Is that true, though?
A I think it's all head cancers, and parotid gland cancers would fall into that category.
Q You're saying there's a known specific association between parotid gland cancers and cigarette smoking?
A. I believe so. I guess I haven't specifically looked at parotid, but have lumped them as part of head and neck cancers.
Q So you don't know whether vinyl chloride is actually following a metabolic pathway of some type to the parotid gland or not?
A I believe it's through direct contact.
Q With the parotid gland. Is that right?
A Through the duct to the parotid gland, yes.
Q You're just presuming that because it's in the same general area. Is that right?
A Yes.
Q You have not seen any evidence one way or the other?
A No.
Q The second factor that you mentioned was the zymbo gland cancers. I presume these are the ones

arising in the laboratory animal studies. Is that right?

A Yes.

Q And their similarity to humans?

A Salivary parietal glands, yes.

Q Why do you say that?

A Same location, some of the same function in terms of the salivary secretions, and so on.

Q You say zymbo glands have salivary secretions?

A Yes.

Q You also mentioned, however, that humans don't have zymbo glands?

A That's right.

Q What was the prevalence of zymbo gland cancer in the animal studies?

A I don't remember.

Q Was it a high incidence?

A There was dose response, but I don't remember. I don't remember.

Q What study are you referring to specifically?

A Some of the early 1970 or early '70 studies, some of the first toxicologic stuff, data, and I

think some of the stuff that Viola did.

Q These were the exposures at 30 parts per million, do you recall?

A How much?

Q 30,000 parts per million.

A I think so, yes.

Q Do you know what the function of a zymbo gland is?

A It's similar to a salivary.

Q Where do you get that information?

A Physiology.

Q Animal physiology?

A Yes.

Q Do animals also have their own salivary glands?

A Yes.

Q And they're saying they're just duplicative?

A I believe so, yes.

Q The third factor that you mentioned was your experience with oral cancer.

A Yes.

Q This was the previous case that you had had?

A Yes.

Q You said that this gentleman had exposure to

vinyl chloride. Is that right?

A Yes.

Q And he had a mouth cancer?

A Yes.

Q From this one experience, then, you're saying that vinyl chloride causes mouth cancer, head and mouth cancer. Is that right?

A I didn't say that.

Q All right. Well, tell me what factor that plays.

A That plays a factor, and there are other cases as well of oral cancers with vinyl chloride exposure, so that I would say there's a series of case reports that lend credence to cause and effect.

Q What other case reports are you talking about?

A There's an example, I think, of a young kid who chewed on a PVC plastic, for example. There's a couple others that I don't offhand recall.

Q Where did you see these couple of others?

A I can't -- again, if you want, I can produce those for you if you're interested.

Q Well, Doctor, you understand that I'm trying to get the basis for your opinion here.

A I understand. I just don't have them, you know, right here to turn to.

Q Well, if you get them to me later, I can't ask you questions about them.

A All right.

Q So I'm familiar with the one.

A The kid that was chewing, all right.

Q I understand that, and that's a case report that you're relying on. Is that right?

A Yes. And the others were case reports like that, but they weren't chewing. They were exposures to PVC.

Q Involving what now, again?

A Now I forget.

Q Okay. You'll have to provide those to me. Okay?

A All right.

Q These series of case reports, what significance does that have to you, then?

A Well, they become a piece in the puzzle, so to speak. They're not solid epi. proof, but they're one of the problems you deal in when you have rare cancers such as this. It's really hard to get a large enough series, you know, so they become pretty

important.

Q That oral cancer case that you had before, do you remember the name of that gentleman?

A Yes.

Q Can you tell that to me?

A I don't think so. That's confidential.

Q All right. What were his exposures to materials? Was he a smoker?

A I don't believe so.

Q Did he chew?

A No.

Q Did he drink?

A There was some alcohol, I think.

Q He was producing vinyl chloride?

A He was exposed to it, yes.

Q In what way?

A Well, he was involved with the design of, or redesign maybe, some ventilation systems where vinyl chloride was being used and being -- I think they were making PVC resin at the facility. I forget all the details.

Q Was he a regular worker there, or he just came in for this subject?

A He was not a regular worker. He had been in

and out of this facility.

Q Now, the fourth factor that you mentioned was the lack of the two primary causes known to you, these being alcohol and cigarette smoking. Is that right?

A Yes.

Q You've also indicated to me that the majority of these types of cancers, there is no known cause. Is that right?

A That's right.

Q So why does the absence of these two particular causes lead you to the conclusion that it must be vinyl chloride?

A Well, those are the two most common known causes, and given now a third rather significant exposure, I think that leads me to say that the vinyl chloride is the most likely cause here.

Q As opposed to the cause of all these majority of --

A Being unknown, that's right, because we have something known here.

Q So you're pointing to vinyl chloride because you know its there. You don't know what caused these other cancers. Is that right?

A Yes. I mean, it's more complicated than that, of course. In lots of head/neck cancers not only don't you have -- you don't have any data at all. Okay. You have a death certificate and no ability to get any information, and that forms, then, the basis for unknown cause or etiology.

Q I'm sorry, Doctor, but isn't that a bootstrap argument, saying that it's vinyl chloride that caused this because he had exposure to vinyl chloride?

A I guess I don't understand bootstrap.

Q Well, you have all these other unknown causes. How can you distinguish this particular case from being subject to some other known cause?

A Well, if I didn't have other information about vinyl chloride, if vinyl chloride had never been shown to be carcinogenic, didn't know its ability to be mutagenic, didn't know about the laboratory data in terms of animals, yes, it would be pretty tough.

Q Okay. So this alone would not permit you to conclude?

A That's right, that's right.

Q The three case reports alone would not allow

you to conclude that vinyl chloride was the cause.

A That's right.

Q Is that right?

A That's right.

Q The animal study alone would not allow you to conclude that this was the cause. Is that right?

A That's right. There's another piece of this puzzle, and this is really true for both of them. I don't mean to skip around, but both had X ray evidence of interstitial fibrosis. I didn't see that it was well worked up, but that also I put in the interesting category. Vinyl chloride is associated with, and PVC, both are associated with a pneumoconiosis, some interstitial fibrosis.

So this piece of evidence certainly by itself doesn't mean a lot, but in the total picture is another piece indicating some exposure. And, of course, the inhalation route was the means of exposure, the primary means of exposure.

Q You're saying that the interstitial fibrosis that you saw reported in the medical records you believe to be due to the vinyl chloride exposure as well?

A No, I'm not saying that. I'm putting it in

the interesting category. Okay. Vinyl chloride and PVC are associated with those types of processes.

Q Now, you also mentioned as a fifth factor the unusual nature of this cancer. Is that right?

A Yes.

Q How does that lead you to believe that vinyl chloride was the cause of it?

A Because that's an unusual exposure and an unusual cancer. Not many people have vinyl chloride exposure. Not many people work with it. That, again, of itself doesn't stand all on its own, but it's a part of a piece, a piece at least, of the whole picture that tips that medical evidence scale in terms of probability.

Q Doctor, have you looked at the epidemiologic evidence for head and neck cancers among those exposed to vinyl chloride?

A I've certainly looked at -- I don't think so. I've looked at epidemiologic evidence for head/neck cancers, yes. In terms of the cancer rate in the cohorts with VC exposure, I think the cancer is rare enough that it has been hard enough to give you much cause and effect there.

Q Several of the studies have had fairly large

cohorts, over a few thousand people. Is that right?

A But small numbers of head and neck cancers and particularly this type is the problem.

Q You have not seen any excess rates of cancer among those exposed to vinyl chloride in those cohorts, have you?

A I don't believe so.

Q You have negative epidemiologic evidence, then, of any association. Is that right?

A Well, I take that back. I think there is part of a basis for some of those -- the case report is an epi. report. I don't immediately recall where it is or who wrote it or what, but that's something I can get to you if you're interested.

Q What case report?

A Well, the case reports of oral cancer from vinyl chloride.

Q No. I'm talking about real epidemiological studies.

A I understand what you're talking about, and I'm saying part of the basis for those was an epi. study and I can find that for you if you're interested.

Q I don't understand what you're saying. Part

of the basis of what was an epidemiological study?

A Part of the basis for the oral cancer in the vinyl chloride exposed individuals, the two or three case reports, was an epi. study showing head and neck cancers, and I will look for that for you.

Q That is part of the basis for your conclusion. Is that right?

A I believe so, yes.

Q Doctor, you're referring to the case report involving the young man that chewed on the wire.

A Yes.

Q Correct?

A Yes.

Q That makes reference to the Tabershaw-Cooper original study. Is that right?

A I believe it does if you have it there to refresh me, yes.

Q I'm happy to do that.

I can't find it here. Do you have it, Doctor?

A Well, I think I do. I don't know whether I have it immediately here or not.

They showed an SMR of 189.

Q Where are you referring to now?

A That Tabershaw study.

Q What is the date of the study, sir?

A '74.

Q Did you see the follow-up after they had accumulated all the information published by Clark Cooper in 1981?

A I may have.

Q Well, let me show it to you.

A Okay. Entitled "Epidemiologic Study of Vinyl Chloride Mortality through December 31, 1982," by Clark Cooper.

MR. DELLI BOVI: What was the date on that, again?

MR. BUNDA: October 1981, Volume 41, "Environmental Health Perspectives."

Q Do you see where the SMR was 100? In a follow-up in 1978 the SMR was 100. Two-five were expected and 5.19 were found.

A Yes.

Q Do you see that?

A Yes.

Q That doesn't give any support to any claim that there was an association between a vinyl chloride and an excess number of people?

A Well, I would want to look at that.
Q I understand that, Doctor.
A But on the surface that's right. An SMR of 100 is not supportive.
Q Are you aware of any other epidemiologic studies that support your conclusion with regard to an association between buccal cavity cancers or head and neck cancers and vinyl chloride?
A Not offhand.
Q Doctor, we talked before about the relative strength of various types of medical information or medical articles, and at that time you agreed with me that there was a hierarchy and that epidemiologic studies, if they were well done, were more significant in terms of their information than, for example, case reports. Is that right?
A Yes.
Q And the negative findings in an epidemiologic study would, in your opinion, be more significant than your case reports that you found?
A Depending upon the strength of that negative study, they could be, yes.
Q And for that matter, the animal studies that we've talked about, we've acknowledged there are

problems in trying to associate effects in animals with possible effects in humans because of the difference between animals and humans. Right?

A That's true.

Q If there is an effect in an animal study, that may or may not be indicative of what would happen in humans, right?

A That's true.

Q And in fact, there are effects which are found in animals which are not found in humans. Is that right?

A That's true.

Q In the particular situation of your buccal cancers, that doesn't seem to be following through with the human data that we have through the epidemiological studies that we have, does it?

A Well, again, I want to see the follow-up study. I mean, there are several things that are quite interesting in this, the fact that buccal cavity cancers occurred early with less exposure, less duration of exposure.

Q Those are all indicative of the fact that vinyl chloride did not have an association?

A Not necessarily at all. It may, in fact,

relate to a specific mechanism for buccal cavity cancer.

Q You're indicating to me, then, that having less of an exposure rather than more of an exposure, having a shorter period of time of exposure, having an earlier exposure, is more indicative of cancer than those that don't get it when having the long exposure, more of an exposure and at a later date?

A I think you misunderstood what I was at least trying to say.

Q I hope so.

A It may, in fact, speak to some of the peculiarities of exposure, the job. And you need to look at what other factors as well when you see something like that.

Q It may also speak to the fact that it's just a coincidence?

A That's true.

Q Are you familiar with the study which has been recently published by NIOSH studying Union Carbide workers in West Virginia?

A Yes.

Q That does not support your opinion with regard to an association between buccal cancers and

vinyl chloride, does it? I'll refer you to page 436 of the report, Doctor.

A It does not.

Q Now, you've indicated that the evidence supporting the association or causal relationship between vinyl chloride and Mr. Dendinger's cancer is less strong than that which exists for Mr. Wallace. Is that right?

A I think so.

Q The factors that you mentioned supporting your opinion with regard to Mr. Dendinger are, number one, the fact that there's no strong social causes that you can find. There's a mechanism there for vinyl chloride to get to the colon area.

A Yes.

Q And the epidemiologic study from this particular plant. Is that right?

A Yes. And some of the low levels but nevertheless increased risks from several other epidemiologic studies, although certainly not all.

Q I'm sorry. I didn't understand that all.

A Evidence of several epi. studies of some positive association, although it's not present in all studies.

Q What are those studies that you're referring to?

A Which I can certainly get that to you if you're interested.

Q You're talking about the Chiazze study?

A I think that's one.

Q The Georgetown study?

A I don't know it by --

Q That's the same thing.

A Okay.

Q Anything else?

A I can get them to you.

Q I want them now if I can get them.

MR. DELLI BOVI: Well, let's take the time and we'll go through them.

MR. BUNDA: All right. Let's go off the record for a minute.

(Recess taken.)

THE WITNESS: I'm ready.

Q What was the article?

A This is a list of quite a few here.

Q All right. Tell me.

A The British Journal of Industrial Medicine, 1984, 41:25-30, Heldaas. JOM, October 1981.

Q Hold on just a second.

Okay. The second one?

A JOM, October 1981, Volume 23, Number 10, Therialt, T-h-e-r-i-a-l-t.

JOM, the Chiazze article, Volume 19, September 1977.

Chiazze's article, Environmental Health Perspectives, Volume 41, page 187-190.

Tabershaw's article, JOM, August 1974, Volume 16, Number 8.

Journal of Cancer Research and Clinical Oncology, 1981, 102, pages 1 to 11, by Emmerich.

Environmental Health Perspectives, Volume 41, page 141 to 151, 1981, Molina.

Environmental Health Perspectives, Volume 41, pages 95 to 99, 1981, Weber.

Viola's reports. I'll have to dig out some of these. These are another article. I'll have to dig out which ones.

Archives of Environmental Health, Volume 30, July '75, page 333, Ott.

The Lancet, January 31, 1976, Baxter, page 245.

Annals of New York Academy of Science,

Volume 246, page 225, 1975, Nicholson.

JOM. Oh, Tabershaw's article. Never mind.

Lancet, August 17th, 1974, Monson.

Industrial Hazards of Plastics and Synthetic Elastomers, pages 155 to 175, and it's from -- actually it's from Progress in Clinical Biological Research, page 155 to 175. 1984, Volume 141, pages 155 to 175.

And I guess it's some of Viola's, a review of initial work published in the Italian journals. What are they? Medlav. I forget the name of them. 61, 174 to 180, and 65, 81 to 89.

Q What are you referring to there on this last one?

A Viola's. It's from this German article.

Q All right. Let's take them one at a time. You're referring to the Heldaas article as number one?

A Yes, okay.

Q What is it in there you find to be significant with regard to colon cancer?

A Table 3 showing increased under ICD code 153 is colon cancer, increased observed expected ratios.

Q They saw three and they expected to see

1.44. Is that right?

A Right. Well, no. They saw three and expected to see .92.

Q Did they find whether that was statistically significant or not?

A I don't see statistical analyses, but that's a PMR of 3.30.

Q I understand.

A Well, anyway, the relative risk is 3.3.

Q I understand that. But is that statistically significant?

A I don't know statistically.

Q These were in a high exposure group. Is that right?

A Yes.

Q Do you know how they designed the exposures?

A They were based upon sporadic measures of explosion meters and some estimates of odor thresholds. And then based upon time of employment, categories were made, although there were nine exposure categories.

Q The other two exposure groups showed no basis. Is that right?

A That's right.

Q Do you know where Mr. Wallace or Mr. Dendinger would have fit in those exposure categories?

A I really don't, because I don't have the actual measurements. If you believe his ability was correct in detecting vinyl chloride, then he would have been in the higher category. If my estimates are more of a one to five parts per million with peaks of 25 to 100, then he would not have been in that higher category.

Q You don't know whether that was statistically significant or not, do you?

A I don't know, but you have a relative risk over two with a dose response from low to high.

Q You understand that relative risk means nothing if it's not statistically significant?

A That's right, although relative risks of two or greater are thought empirically to be of some significance.

Q If there are enough people in the cohort to make the statistics valuable?

A That's correct.

Q And there's a question here as to whether three is statistically significant enough to make it valuable?

A That's correct.

Q All right. What next do you find to be important?

A What was the next article? The Therialt, the Journal of -- JOM.

Q JOM. What do you find to be important there?

A Table 4, relative risk, 6.25, statistically significant.

Q That's of all digestive cancers. Is that right?

A That's correct.

Q Does that include the liver cancers? Well, I direct your attention, Doctor, to page 674, the left, the text down there on the left-hand side. Do you see the language that says: As for specific cancer deaths, a significant excess is noted for cancer of the digestive system only?

A Yes.

Q Then would you read that next sentence?

A The excess is accounted for exclusively by cancers of the liver.

Q No other cancers are in excess. Do you see that?

A Yes.

Q Does that have any significance to you?

A Well, yes.

Q That would indicate that this does not support any association between colon cancer and VC. Is that right?

A Well, I wouldn't say that it doesn't, but the liver --

Q It doesn't lend support to it?

A That's right.

Q Thank you. So we can cross that one off. What's the next article? The Chiazze article, right, for 1977?

A Yes.

Q You mentioned that one, and the one after it is a Chiazze article of 1981. They're two publications from the same study. Is that right?

A I believe so.

Q Well, satisfy yourself. That's my understanding.

A The one from '77 shows a PMR, elevated PMR.

Q That's a proportionate mortality study. Is that right, Doctor?

A That's right, with cancer of the intestines

for both men and women being increased. The other Chiazze article is October '81. Although published afterwards, it just lends more support to the cancers of the digestive system.

Q What do you mean it lends more support?

A Well, they've done some more. A case control analysis has been done.

Q That was on a breast cancer. Is that right?

A Yes.

Q Because they also found an elevated proportionate mortality ratio for breast cancer, right?

A Yes.

Q That didn't pan out when they did a case control study, did it?

A That's right.

Q In other words, they compared it to the exposures that the people had, and the people with the lowest exposures had the highest rates of cancer?

A But in terms of the large intestine they've got statistical significance now here.

Q Well, they had statistical significance with the breast cancer with regard to the proportionate mortality study, didn't they?

A Yes.

Q And when they checked it more thoroughly with a --

A Case control.

Q -- it didn't carry through?

A But the large intestine with additional data was still valid subsequently.

Q Well, Doctor, listen to my question. They had statistical significance, in light of what they were looking for in the study, for both breast cancer and colon cancer. Right?

MR. DELLI BOVI: You're talking about the '77 study?

MR. BUNDA: Well, they're both derived from the same data.

A Yes.

Q So the answer to my question is yes?

A Yes.

Q We're already talked about some of the problems with the proportionate mortality study. Correct?

A That's right.

Q So the next step, then, is to go to a case control study where you look at the actual exposures of those people. Correct?

A That's right.
Q They did that for breast cancer. Correct?
A That's right.
Q And that contradicted what they thought they would find from just looking at the proportionate mortality data. Is that right?
A That's right.
Q Which weakens the significance of the proportionate mortality data that they found it indicates?
A For breast cancer only.
Q Well, they didn't do the comparisons for the colon cancer, did they?
A That's right.
Q So you don't know whether they're going to get the same result that they did with the breast cancer, that is, disproving it or not, do you?
A You're right.
Q Which points out the shortcomings of a mortality study?
A That's right.
Q You can't use that to establish a cause and effect relationship, can you?
A No, that's not true. PMR studies can be

quite helpful. By themselves, no, but with the accumulation of other data, definitely lend credence to relationships.

Q Do you understand that a proportionate mortality study is only appropriately used as a screening device?

A Yes.

Q To determine further investigations?

A Yes.

Q And you can't use that to establish cause and effect?

A But that's not what I just said.

Q I understand. But you agree with me that you can't use it --

A It's a screening. It by itself should not be used exclusively for cause and effect, that's right.

Q You have to do further investigations to confirm what is suggested by these studies before you can establish what is cause and effect even as suggested by a proportionate mortality study?

A You would like to use that.

Q And the data on a further study, then you accept that data if on further and expanded epidemi-

ological studies it disproves that there is an association?

A It can be done.

Q Have you seen the expanded data?

A I don't think so on this particular --

Q I understand that. If you are presented with expanded data on an epidemiologic study of PVC workers which disproves an association, then you would have to accept that?

A If the study is right, yes. I mean, if it's done well, and so on.

Q So based on this information alone, you would agree with me that the Chiazze articles are not sufficient for you to say Mr. Dendinger's cancer was caused by vinyl chloride?

A That's right.

Q The next is the Tabershaw article, you say?

A Yes, of seventy--

Q Of '74?

A Yes.

Q You understand that there was a follow-up to that article or an expansion, a further collection of data?

A I understand.

Q If we look at that, that's a more complete study than the '74 study?

A Although there are real problems with inclusion in the study, and so on. It seems -- I haven't looked carefully at the follow-up to see -- they excluded a number of people in this first study that would have increased even the relationship, I think, between colon cancer and vinyl chloride.

Q Well, what are you talking about? Point me to what you're referring to.

A Okay.

MR. DELLI BOVI: Is this what you're looking for?

THE WITNESS: Yes.

MR. DELLI BOVI: I don't know if he wants you to use my copy or not.

A (Continuing) Yes. Did the follow-up study, for example, continue analysis after the end of the cutoff? I haven't looked at that to know.

Q Well, I thought you were saying, Doctor, the 1974 study, they significantly excluded people which otherwise would have increased the colon cancer rate.

A Yes.

Q Where is that reference?

A The cutoff date was December 1972. For example, some individuals in one particular study were -- records were included of individuals who were terminated only in the six years prior to 1972 instead of looking through it for everyone that had ever worked at the facility. So I --

Anyway, I haven't looked at the follow-up article to know what, in fact, they did.

Q But you agree with me that there is nothing in the '74 article which indicates that the people which they excluded, if they had been included, would have increased the colon cancer rate?

A Well, you would have expected it, that, in fact, it might have, because these were individuals that had worked longer, had longer latency periods, had been farther away from exposure in terms of time. So that, since that's the group that you would expect to have a problem, may have, in fact, contributed or added to the risks that are the -- and the data that's presented here in '74.

Q But in fairness, Doctor, you don't know what they would have done to the group?

A Oh, that's right.

Q So you're guessing that they may have or

they may not have. We really don't know?

A Well, epidemiologically, as you critique a study, that's a bias, you would say, which would tend to underestimate the risk.

Q That's a bias. That's a problem with the methodology of the study, but you don't know what it would have done to the results of the study?

A Well, yes, that's right.

Q Well, let's look at the follow-up, then, in 1982 to the Cooper study. You were asking about what the follow-up did.

A Yes.

MR. DELLI BOVI: Do you want to use my copy?

THE WITNESS: Yes.

Q Do you see on page 102 they said that 85 percent of the study population was located in the first study? In the follow-up they raised that level to 95 percent. That's under the heading "Follow-Up."

A All right. Just a minute. Let me look at this, please.

Well, clearly there continues to be a trend for excess digestive cancers even in this study, and it is true they did include more individuals and go back farther and could account better in terms of

total exposures and latencies, and so on.

Q Where do you see the support in this report for saying that clearly there's a continued trend for increased digestive?

A In the abstract on page 103 under the SMR.

Q The digestive tract includes the liver cancers. Is that right?

A That was not my understanding that it was.

Q Well, look at the numbers indicating after each one of the cancers that indicates the, what is it, the international index.

A The ICD code.

Q Yes.

A 150 to 159.

Q That includes liver cancer, doesn't it?

A Well, as a matter of fact, I understand the angiosarcoma deaths that were part of this were not coded -- only four of them were coded as tumors of the digestive tract, but they don't specifically -- I don't see talk of how the angiosarcomas separate out and what effect they have. They conclude there continues to be a slight but inconclusive trend for higher SMRs for deaths in digestive tract and inspiratory tract tumor, and certainly as you look at

low/medium/high data, less than 20/greater than 20 years' exposure, you see trends there as well in terms of increasing rates, risks.

Q You're looking at?

A Tables 5 and 6, for example.

Q Tables 5 and 6. And even in the high exposure the SMR is --

A 117.

Q Is that statistically significant from the norm?

A Well, in this situation you need to compare it to low and medium and to see what's happening over time, and that's certainly different than the SMRs from the low and medium group.

Q Where would you place Mr. Dendinger in this? Would he have a low, medium or high exposure?

A Well, we talked about this. I mean, if, in fact, he's right that he could smell it, then he's in a high group.

Q And if he's wrong that he could smell it?

A Then he's in a lower group.

Q And, in fact, according to this, he would have a lower than average rate?

A Well, now you're taking -- okay. You're

trying to -- the man has cancer, so I mean, it's not a question of whether or not he was going to get it. His risk for getting it if he was in a low group may have been lower but, in fact, he has cancer.

Q Well, let me back up for a second. If we're trying to evaluate what caused the cancer, those people that had colon cancer and were exposed to low or medium levels of vinyl chloride were getting cancer at a lesser rate than the normal population was.

A Yes.

Q Which does not indicate any association.

A Well, we don't know what it means.

Q Well, if they're getting less cancer than the people outside the plant, that would seem to indicate that there's no association. Is that right?

A Right. However, but then you look at dose response, and if you see higher rates and higher exposed, then you know even the cancers in the lower exposed group may, in fact, be related to exposure, but you see dose response. Do you understand what I'm saying?

Q No.

A If, in fact, the only data we had was in

this low group where there was the SMR was less than the normal, you're right, you can't talk about association. But if you have dose response demonstrated across exposures, showing increasing rates across exposure, then you can no longer say that those exposures in the lower groups did not even cause those. You've show an association that occurs with exposure. You don't know where -- we're back to the threshold argument. You don't know where a no threshold level is, and you're hard pressed to say that exposures did not cause those cancers in that low group even though they're the less than the, quote, unquote, normal population.

Q Have you ever heard of something called a healthy work career effect?

A Yes.

Q Tell me what that is.

A People that work are healthy by definition. They're able to go to work. They're healthy enough to get out of the house, do their job, perform it well. So by definition, people that work are going to be healthier than those who don't.

Q And people who work longer are going to be less healthy as time goes on. Is that right?

A That's right.
Q And the longer they work?
A It speaks something to work, doesn't it?
Q It also means that just through the aging process you're going to get sicker from time. You're not going to be as healthy as you once were.
A Yes. What happens when the healthy worker catches up to the rest of the population isn't real clear. There are several things you can say. One is that the work may have caught them up and is making them no longer healthy enough to work. Or you can say that there's something peculiar to the aging of workers that's different than the aging population in people that aren't working that makes them catch up to people all of a sudden. It's pretty tough to figure out what happens when the worker catches up to the nonworker.
Q And that would be an explanation as well for what's happening here?
A No, I don't think so.
Q Equally as well as your exposure explanation?
A No. Because we're not talking about -- there's nothing on Table 5, there's nothing about

length of exposure or age or anything else that has to do with dose.

Now, the next group in terms of exposure and length of exposure, that's where you could try and make that argument that it's just that they're catching up to the nonworking population. That doesn't fly very well, I think, when you look at separating in terms of exposure time.

Q The conclusion of the authors was that?

A A slight but inconclusive trend.

Q All right. So they didn't draw any conclusions from the trend. Is that right? That's what the word inconclusive means?

A I think they're saying that statistically they haven't excluded chance, but the trend that has been reported before and elsewhere continues with this data.

Q You also see where they suggest later down in that paragraph, "The results suggest that except for a proven association with hepatic, h-e-p-a-t-i-c, angiosarcoma and a strongly suggestive association with central nervous system tumors, vinyl chloride probably is not associated with significant excess cancers of other sites."

Do you see that?

A I see that.

Q You're disagreeing with their conclusion. Is that right?

A I think, again, we're showing again a trend coupled with other -- by itself this study doesn't stand, but it continues to support digestive cancers, colonic cancers.

Q In your opinion, this supports colon cancers?

A It lends credence to it.

Q That's not what the authors say. You disagree with the authors. Is that right?

A Well, you know, this is a scientific publication and they're hedging their bets. They talk about the continuation of the trend. I would not say that this by itself proves colon cancer, but it's a piece.

Q If there were a follow-up to that study which disproved that, then you would have to carefully analyze that information?

A I would want to see the follow-up, that's right.

Q The next one is the Tabershaw. No, we

talked about that. Tabershaw for '74 to '81.

A Yes. I've got this Nicholson, the Progress in Clinical Biological Research.

Q What is significant in that?

A Actually, I think this one is just bringing up the Chiazze articles.

Q What is the title of the article?

A It's "Occupational Hazards in the VC-PVC Industry." This is a review article.

Q Dr. Nicholson indicates that if Chiazze had just looked at proportionate cancer mortality ratios, he wouldn't have got a statistically significant number. Is that right?

A He says that if they had, that they would have had elevations, but perhaps not all the way to .05 in terms of significance.

Q Which means it is not statistically --

A Well, now, you can't just rest your head on the P value being .05. That is a relatively arbitrary number, and you know there's still a possibility of chance, one in 20 of chance at P.05. At P.1 there's a one in ten chance. So you need to look at the P value to know exactly what it means. The lower it is, the more striking the relationship or less

likely chance.

Q Well, that's the value that the epidemiologic community has taken which shows you have achieved some significance?

A P.05 is the number, but what's the difference between .05 and .06? There's very little. .06 leads to significance or support. Even up to .1 indicates a trend. So it's important not to just use .05. It's really important to always list all P values so you can have a sense of how close the significance is.

Q But you agree with me that the P value of .05 has achieved some significance?

A By convention, right, we take one in 20 as being appropriate.

Q They have to draw a line someplace, because when you're dealing with numbers like this with large groups of people, you can achieve by chance --

A You're right.

Q -- some indication?

A Yes.

Q And this is an effort to try and minimize that?

A That's right.

Q So what Nicholson is saying, if I can summarize, is that by changing the way these numbers are looked at, they may or may not have significance?

A That's right, that's what he's saying.

Q And that in his opinion, it would have been more appropriate to compare the cancer rates with the cancer rates --

A That's right.

Q -- of the national population?

A Well, he was talking about proportionate in this situation.

Q Well, in a proportionate study, nevertheless, you look at the cancer rates within the population and compare to the national cancer?

A Well, that's a national SMR. All he was doing here was the PMR, but he was suggesting he should look at the proportionate among the cancer deaths and not just all deaths, and that would have, by doing that, reduced the significance. Instead of taking all the deaths, Nicholson is saying look at just the cancer deaths and do a proportion of the cancer deaths alone.

Q I understand that. When you're taking all of this data, whether it be proportionate or a stan-

dard mortality study, and when they by necessity have to compare it to a national standardized rate, you have --

A Well, you always compare cancers to an SMR, and you always compare --

Q Well, let me finish my question.

A I'm sorry.

Q When you're taking that information and attempting to apply it to this situation in Mr. Dendinger's case, you have to evaluate Mr. Dendinger's situation, lifestyle, comunity, to determine whether that community is consistent with the national average. Right?

A That's right.

Q And if, in fact, there's something else in the water or something else going on in that area which causes an increased cancer rate in that population, that may have some significance to you. Correct?

A Yes.

Q Because he may be living in a more dangerous area where there's just more cancers, for whatever reason?

A That's right.

Q You haven't done that?
A That's right. That would be very difficult.
Q As we indicated, those statistics exist, don't they?
A That's right.
Q You haven't done that for head cancer, either, have you?
A No, I haven't.
Q If those rates were elevated, you would have to take that into consideration?
A Yes.
Q The Molina article is the next one.
A Okay. Table 4, digestive tumors increased.
Q Again, do you know whether that includes the angiosarcomas or not?
A That's what I'm looking to see.
Q Look on page 149. Do you see on the second column just above "Discussion" they indicate that a liver cancer is ICD 155?
A Yes. One of the 11 tumors was liver.
Q I understand that. Do you see before that where it states: In the question of tumors of the digestive organs, 11 cases were observed as opposed to an anticipated 8.5?

A That's right.

Q The difference was not statistically verified. Is that right?

A Yes, that's right. Now, that's in the group with six months of exposure. When you go with at least two years, the relative risk does go up and the numbers are smaller.

Q Where are you looking at?

A If you look at Table 8, Table 8 is two years of exposure. The odds ratio is 1.85 as opposed to 1.29. So you see a similar type trend, longer exposure, increased odds ratio, where admittedly the numbers are down so that --

Q It could be chance?

A -- it could be chance. But, again, we're we're building quite a picture here of these various bits and pieces in showing some of the real problems with doing epidemiologic work: you have small cases, lots of missing data, what are other variables, that you need to take several studies like this that really then begin to lend support to cause and effect.

Q How many colon cancers are we talking about?

A Well, there were 11 -- ten, apparently.

Q No. We're talking about digestive cancers in that 11?

A Well, 11 minus one.

Q Well, no. Wait a minute.

A Isn't that what they say?

Q No. You're talking about digestive organ tumors?

A Yes.

Q Which would include stomach. It could include rectal cancer. It could include a lot of things. So you can't tell from this whether they're colon cancer or not, can you?

A That's right; that's right.

Q And if you take out the liver cancer, which we concede there's an association with, then we can even further your claimed trend?

A You're right.

Q And even if we don't, the authors concede that there's no statistical confirmation one way or the other.

A That's right.

Q What is the next one? Weber?

A Okay.

(Recess taken.)

Q Okay. What is significant about this?
A We've got significant SMRs for GI tract cancers, which excluded the liver.
Q Where are you referring?
A Table 2. On Table 3 SMR goes up with exposure of GI tract. In Table 4 exposures prior to '59 were -- no doubt exposures were higher. SMR is higher.
Q Let's look at the exposures after 1970, which is the type of case we've got here.
A In the '70 to '74 period, significance is detected.
Q Anything else?
A An interesting one in terms of SMRs by age is showing significance in the group 35 to 44. I think that probably -- well, you'd have to look at that to see what that reflected. Let's see. Probably a reflection of exposure and timing of exposure.
Q So what you're presuming is as the exposure and the time goes up, then the rates of cancer will go up?
A Yes.
Q And if the exposures are lower, then the chances are less?

A That's right.
Q Could you look at your copy of that again, please.
A Yes.
Q I think you're ignoring a statistic there that I would like you to look at in Table 2.
A Okay.
Q Do you see the column labeled "PVC Processing"?
A Yes.
Q What is the standard mortality ratio there?
A Does that say 56?
Q It does to me.
A Okay. It's blurred on mine. I can't see what it is.
Q Well, mine says 56. Do you want to look at mine?
A Okay. I believe you.
Q What kind of people do we have involved in this case? Mr. Dendinger was involved in PVC processing work.
A That's right.
Q So that would tend to confirm -- well, that would not establish an association with PVC process-

ing, would it?

A By itself, again, if we're looking at the overall -- I mean, we're back to the dose response problem here, and that falls in with those responses.

Q So we're seeing a dose response relationship here?

A That's right.

Q You refer next to a Viola report.

A Yes. I don't have that quite handy, but that's the initial, when he went back and reviewed the laboratory data and saw digestive tract cancers.

Q In what, his animals?

A In his animals, yes.

Q Was that his published article or --

A I believe so, yes. It's in this German article, is what they refer to. Page 23.

Q In the German article?

A Yes.

Q Who is the author of that?

A Oh, and halfway down -- oh, it's this one (indicating). The Advances in Internal Medicine Pediatrics. This one. Oh, I'm sorry. Frick.

Q Just a minute here. I know I've got it. What page?

A 23. About halfway down the page after Keplinger, on re-examining histologic slides of his past experience, also detected angiosarcoma of the liver and other malignancies of skin, lungs and intestines of his rats.

Q Did you ever see that in the Viola articles themselves?

A I don't know that I specifically have those articles. And in the references that's in the Italian, the Medlav, '70 or '74.

Q Well, Tony doesn't show that, does he, when he re-examined the tissue slides from those annals?

A I don't see where it specifically says whether he did or didn't disagree with Viola.

Q Other than that reference -- and that's a secondary reference. Is that right?

A Yes.

Q You don't have the original Viola article?

A No, no. Or I may, but I didn't --

Q Well, do you or don't you?

MR. DELLI BOVI: You mean in this room?

MR. BUNDA: I mean, does he have the Viola article that he's referring to?

MR. DELLI BOVI: Yes. In his possession

now?

MR. BUNDA: Yes.

MR. DELLI BOVI: Oh, all right.

A I'm looking to see whether I have it in my possession. I think I just have the secondary.

Q This also talks about other animal studies. Is that right?

A Yes.

Q Has anybody else succeeded in replicating the induction of intestinal cancers in animals?

A I don't know. I haven't seen it.

Q You haven't seen any reference in this particular volume or anything else that you've seen?

A No.

Q And what do you have, a couple of inches of materials before you?

A Yes.

Q So if it had been replicated, generally you've looked at a lot of material to look for it?

A I've looked at a lot of material, yes.

Q What is the next significant article?

A Well, there's the Lancet article by Monson. Digestive odds ratio of 1.6. Just one of the typical short reports in Lancet.

Q Does that have any significance to Dr. Monson in the article that you can see?

A I think basically what is reported here is just the multisystem cancer. It doesn't specifically talk about the GI cancer.

Q Backing up for just a second, the German article that you looked at didn't mention an association between vinyl chloride and parotid or head cancers, did it?

A I don't think so, although the Viola articles even mention specifically the -- that are referred to in that article mention the mucoepidermoid type tumors seen.

Q Where were those seen, though?

A In the laboratory animals.

Q Where in the animal?

A In the zymbo.

Q It was on the skin?

A And in the mouth, I believe.

Q Well, show me where it says in the mouth, because I've seen it on the skin.

A All right. "The cutaneous tumors, which always appeared in the area in which submaxillary and parotid glands are located, have been histologically

recognized as epidermoid carcinomas, papillomas and mucoepidermoid carcinomas."

Q Cutaneous tumors are tumors on the skin?

A Yes.

Q That's indicating on the area of the skin where the --

A Where the parotid tumor occurs, yes.

Q Well, it's not a parotid tumor; it's the area of the skin beneath which the tumors are located. Is that right?

A That's right.

Q There is not any indication in the article that the tumors were found in those particular glands?

A I believe you're right.

Q By the way, as I'm backtracking here for just second, the Cooper article, do you have that there? That was the sequel to the 1974 Tabershaw study.

A Okay.

Q Oh, I think we've already touched on that. I wanted to ask you about buccal cancers, but I think we've already addressed the fact that there was no increase found there.

What about the Chiazze article of 1978, or 1977 for that matter? Look in the mouth, of mouth cancers, buccal cancers. Do you see the standard mortality ratio for buccal cancers?

A The PMR.

Q Okay.

A Not elevated.

Q What is it?

A .83.

Q So that's below expected. Is that right?

A Yes, in this PMR.

MR. DELLI BOVI: Whose table are you on?

THE WITNESS: Table 4 in the Environmental Health Perspective.

MR. DELLI BOVI: You're looking at the '81 Chiazze?

THE WITNESS: Yes.

A (Continuing) Now, in the '77 article, for females the PMR is increased.

Q Well, we're not dealing with female here, are we?

A Well, we're dealing with small numbers and a PMR, which I think there's other supportive stuff.

Q Well, we can't ignore negative information,

though, can we, Doctor?

A Well, it depends on the type.

Q Well, epidemiologic evidence among people using PVC in the manner in which these gentlemen were using it, I understand, but, of course, mortality studies have their faults, so if you will concede that we can't use that for colon cancer and I'll concede that we can't use it for buccal cancer.

Okay. Don't want to do that.

MR. DELLI BOVI: Maybe we should let the jury decide that.

A I think, you know, you need to look at each situation in the realm of the total picture.

Q You will concede that this is information that you'll have to take into account in considering your opinion?

A Oh, yes.

Q Your Lancet article is the next one, I believe.

A This one?

Q Yes.

A We just did it. This is again showing --

Q I'm sorry. Who is the author on that?

A This is Baxter, and it's showing what is

listed as stomach cancer with an excess cancer rate of statistical significance.

Q That's different than the colon cancer, isn't it, Doctor?

A I believe so, because they have ICD code 151, which is just stomach.

Q 153 is colon?

A Yes, I think you're right.

Q You haven't seen any other articles indicating any increase in stomach cancer, either, have you?

A No. I think that's the only one.

Q The next one?

A Ott.

Q All right. I don't have that one.

A Okay. Archives of Environmental Health. I think this is more just case reports listing several cases of colon cancer.

Q Those are PVC manufacturing employees?

A Yes, yes.

Q The next one?

A Is the Emmerich, Journal of Cancer Research and Clinical Oncology.

Q I'm sorry. What is it?

A Journal of Cancer Research and Clinical

Oncology.

Q And author of it?

A Emmerich.

Q E-m--

A E-m-m-e-r-i-c-h. It's a review of studies supporting increased risks of GI cancers. They report a study from Germany, R-e-i-n-a-l, Reinal. GI tumors elevated, and one, two, three, four, five, six, seven, eight, eight reviews of GI cancers.

Q Can I see that for a second?

A Sure.

Q I don't have a copy of that.

A Okay.

Q All right. What this is is a review of other studies, isn't it, Doctor?

A Yes.

Q And when you're referring to Table 2, they list other studies that they look at with regard to gastrointestinal cancer.

A That's right.

Q Is that right?

A Yes.

Q They mention the Chiazze article, which we've already discussed. Is that right?

A Yes.
Q They mentioned duct which has a .99 ratio, right?
A Yes.
Q That's not indicative of any problem?
A Without specifically looking at the article, no.
Q Fox and Collier is again .91, below one?
A That's right.
Q And then they mention --
A Reinal.
Q Reinal.
A .1, .3.
Q And Tabershaw and Gaffe, which is .94?
A Yes. And Waxweiller, which was .01.
Q All right. Do you see the summary there?
A Yes.
Q What was the only cancer that they found the data indicated a statistical significance?
A Liver.
Q All right. Can we presume from that that they didn't find the data for gastrointestinal cancer to have achieved statistical significance yet?
A Yes. They conclude that there were

increased instances of lung, GI, CNS lymphomas, but that liver is the only thing that's consistently showing statistical significance.

Q What is your next article, please?

A There is an article by Nicholson, and it's mostly, it's case reportings showing increases of cancer rates overall with some case reportings of colon cancer.

Q What is the title of the article, Doctor?

A "Mortality Experience of a Cohort of Vinyl Chloride-Polyvinyl Chloride Workers." It's from the Annals of the New York Academy of Sciences.

Q This is a collection of case reports. Is that right?

A Well, they looked at 257 individuals.

Q May I see that for a minute?

A Yes.

Q Thank you. Well, Doctor, where specifically in the article are you referring to as something that is significant to you?

A They are merely indicating that -- they indicate 17 percent of the deaths were causally related to vinyl chloride, increased rates of cancer in multiple sites, and then list -- where do they

list -- listing cases, and there's one case of colon cancer here.

Q How many people were there in that group?

A 255.

Q Deaths?

A 255 individuals. Nine cancer deaths. It's a small group.

Q All right. Given the prevalence of colon cancer, that could be by chance alone?

A That's possible, yes.

Q In fact, they didn't ascribe any significance to the colon cancer death?

A That's right. They were talking about significance of cancer deaths overall.

Q You haven't found anything published since that time from Mr. Nicholson that ascribes colon cancer risk to vinyl chloride, have you?

A I haven't.

MR. DELLI BOVI: Excuse me. What was the date on that first one, the article you were just talking about by Nicholson?

THE WITNESS: '75.

MR. DELLI BOVI: Well, he's already cited you a Nicholson article in '84.

THE WITNESS: All right.

Q That does mention colon cancer?

A Okay.

Q And the next one is your Lancet article?

A I think we already talked about that.

Q That's what I thought.

A Yes.

Q I was making reference to the Lancet editorial.

A Yes. I've got that. I'm through my list here.

Q You mentioned something about the industrial hazards of plastics.

A Yes.

Q Was that the German article, or was that something else?

A No. We've been through that one. Nicholson did that one.

Q Doctor, you've had what, three courses in epidemiology?

A Or four, yes.

Q Have you participated in an epidemiology, an analysis of data in the preparation of an epidemiologic study?

A Yes.
Q When?
A The skin cancer study.
Q Well, you're collecting data for someone here at the university?
A Yes.
Q Are you analyzing that data as well?
A Well, I've been involved with the design of the study and will be involved with analysis.
Q You're working under an epidemiologist with that. Is that right?
A That's right, yes.
Q And she guided the design of the study?
A Yes.
Q Would you agree that the opinion of a professional epidemiologist would have to be taken seriously by you in connection with interpretation of articles such as you have mentioned?
A Yes.
Q Would you defer to an opinion of a professional epidemiologist in interpreting that data?
A Not necessarily, but I would certainly listen.
Q You would take that very seriously, wouldn't

you?

A Yes.

Q Because he's in the business of interpreting information like that, and yours is the business of reviewing and treating people for occupational diseases. Is that right?

A That's right.

Q What specifically were the cells that turned cancerous in Mr. Dendinger? I realize they were in the colon, but what type of cancer, what type of cells?

A He had an adeno CA, and the colon, the type of cell in the colon, is glandular, and it was one of those cells.

Q You say it was a glandular cell. Do you know what type of cell?

A Well, it's an adeno cell.

Q Again, with Mr. Dendinger, are you making the same presumption that you did for Mr. Wallace with regard to how the vinyl chloride gets to the site?

A Yes.

Q You don't have any information or research which indicates that actually vinyl chloride reached

the cell in the colon?

A There is actually their labeling experiments with carbon 14, C-14, showing that VCM can make it into the colon.

Q Which studies are these now?

A It's here.

Q You don't specifically remember right now what it was?

A I don't remember which one.

Q Doctor, you, I think, have already testified that there is a large incidence of colon cancer in the population. Is that right?

A Yes.

Q And there is no distinguishing characteristic between what you believe to be a vinyl chloride caused colon cancer and another colon cancer which was not caused by that?

A That's right.

Q The statistics that you have seen in the literature which leads you to believe that this colon cancer was caused by vinyl chloride, what is your sense of the increased incidence of colon cancers in those that you've seen? Do you understand what I'm saying? Attributable to vinyl chloride.

A Yes. There's a mild increased risk. The risk is not doubled.

Q Okay. Thank you. I asked you, I think, about the NIOSH study and one of the cancers. I guess I'll ask you about the other. The Union Carbide study that was done by NIOSH, did that show an increase of either colon cancer or head cancer?

A I don't believe so.

Q Do you want mine?

A Yes. Wait a minute.

MR. DELLI BOVI: I'm going to object to the question, but you go ahead and answer.

MR. BUNDA: What's the basis?

MR. DELLI BOVI: Because there's no indication that the study was done only by NIOSH. It appears to me that it was done jointly by NIOSH and Union Carbide. I suppose that awaits the evidence.

MR. BUNDA: Well, I'll revise my question.

Q Whoever did the study, does the study indicate that there's an increase in either of those two types of cancer?

A No.

Q Thank you. Have you looked at the IARC

publication on vinyl chloride at all?

A I'm not sure that I have.

Q You know what IARC is, don't you?

A Yes, yes.

Q The International Association of Research on Cancer?

A Agency for Research on Cancer, yes.

Q All right. I stand corrected. They had a publication on various different carcinogens, and vinyl chloride is one of them. Is that right?

A Yes.

Q Is there any particular reason why you didn't go to that?

A No.

Q All right. They have committees that analyze all the data and all the publications on a particular carcinogen. Is that correct?

A Yes.

Q And they have experts on those committees?

A I believe so. I assume so, yes.

Q Would you place faith in their conclusions regarding the strength and meaning of those, the literature outstanding on a particular carcinogen?

A I don't have fault, I don't think, with

things that I've read from them, no.

MR. BUNDA: Let's just take a break, and I'll see if I can wind this up.

MR. DELLI BOVI: Okay.

THE WITNESS: Yes. I have to get to the hospital yet.

MR. DELLI BOVI: I understand.

MR. MEYER: Will it be any problem if I proceed with just a couple of questions while you're taking your break?

MR. BUNDA: No. Go ahead.

BY MR. MEYER:

Q I take it with your review of the medical records and the background of these two individuals you became familiar with some things about their work history at Chrysler.

A Yes.

Q And particularly familiar with the number of years that each of these individuals worked at Chrysler?

A Yes.

Q Did I understand correctly before your testimony that if you were satisfied that the residual vinyl chloride monomer exposure level for

each of these individuals was consistently one part per million or less that you would consider that exposure a minimal cancer risk?

A Yes, certainly far less than what I think happened. That's right.

Q Given that you're familiar with the total number of years that they worked at the plant and given your opinion there as to the one part per million exposure, if that indeed was so, I would like you to assume a couple of things.

Assume that a company supplied polyvinyl chloride resin for a total of four years and assume that that resin had a residual vinyl chloride monomer content of one part per million or less. Would that particular brand of resin have been a substantial contributing factor to the cancer of either of these gentlemen?

A You know, as I think I said, that if, in fact, the total exposures was a part per million or less, the risk is pretty low. If you've got one particular supplier that all of their resin was one part per million or less, then that contribution would not be great, either.

MR. MEYER: Thank you.

Are you ready to jump back in?

MR. BUNDA: Yes, for just a couple questions.

BY MR. BUNDA:

Q Doctor, in your opinion, what kinds of cancer are caused by exposure to polyvinyl chloride?

A Angiosarcoma, lung cancer, CNS cancer, probably.

Q If I can stop you there. Central nervous system, is there any particular spot? I mean, the CNS system is a pretty big one.

A Well, you refer to the brain. CNS refers to the brain. I believe the hematolytic and lymphocytic cancers and the two particular types here, intestinal, oral cavity.

Q Anything else?

A I think that's it.

Q Well, you've reviewed the literature?

A Yes.

Q So you've pretty much formed your opinion on this?

A Yes.

Q We were talking before about the metabolic pathways, and you were talking about a metabolite

that was created by the body when the cells were exposed to vinyl chloride. How is that metabolite created, when you have vinyl chloride presumably going to the product end, to the parotid gland or the colon?

A The liver is not the only site, and many cells do possess the ability to metabolize these chemicals, possess the cytochrome P450 system. That presumably is how malignancies in other sites than the liver occur, because the life of the intermediate is not long, and I don't think that there's evidence that it's transported from one site to another in the intermediate and most hazardous form.

Q Well, are these cells in the colon?

A Yes.

Q Are these cells in the parotid gland?

A Yes.

Q Is there evidence that they create the metabolite there?

A I think so. That's the presumed. I'm not sure actually that someone has seen the metabolite in any other area than liver homogenates.

Q Doctor, with regard to colon cancer, we've already established that there is colon cancer where

there is no known cause of that cancer. Correct?

A I'm sorry. I lost you.

Q Okay. Well, let me backtrack, then. For every colon cancer there's not always known to be a cause?

A That's right.

Q In the development of some colon cancer, isn't it a fact that the body's own defense mechanisms, the aging of the body, may give rise to the fact that the cancer develops?

A That may be true, yes.

Q So that it is not necessarily true that for every colon cancer there has to be, for example, an exposure to a chemical or to some outside agent?

A That may be true. It's not clear whether there truly are, quote, unquote, spontaneous DNA changes. I have not seen anyone show evidence that that happens.

Q One way or the other?

A All of the changes have been shown to occur with a chemical or substance causing an alteration. I have not seen anyone demonstrate that these changes occur without an initiator, without some specific substance causing that.

Q Well, now, wait a minute. Isn't there established that there could be a virus that could cause cancer?

A Well, that's a substance.

Q Or the substance can be produced within the body itself?

A I haven't seen that shown.

Q There are theories that that is occurring. Is that right?

A I think only theories, yes.

Q But there are? My question is, there are theories that that --

A Yes, yes.

Q In fact, the whole area of the development of cancer is still a very much open question and subject to theories?

A That's true.

Q And the same questions would be addressed to the parotid gland cancer, that it is not necessarily known that only external chemicals cause parotid gland cancers?

A That's true, but, again, the same thing to say that's the only setting, in fact, where the changes have been observed as with the chemical or

substance causing a problem causing the DNA alteration.

Q I believe you already said that in a lot of them we just don't know what caused it.

A That's right.

MR. BUNDA: That's all. Thank you. Signature?

THE WITNESS: Signature.

MR. DELLI BOVI: We'll reserve signature.

AND FURTHER DEPONENT SAITH NOT.

_____
Deponent

C E R T I F I C A T E

STATE OF OHIO :
: SS
COUNTY OF HAMILTON :

I, Luke T. Lavin, a Notary Public duly commissioned and qualified in and for the State of Ohio, do hereby certify that there came before me on the 6th day of May, 1988, at 11:05 a.m., at Suite 106, 2450 Kipling Avenue, Cincinnati, Ohio, the following named person, to-wit, Ralph Michael Kelly, M.D. who was by me duly sworn to testify to the truth and nothing but the truth of his knowledge touching and concerning the matters in controversy in this cause; that he was thereupon carefully examined upon his oath and his examination reduced to stenotypy by me; that the deposition is a true record of the testimony given by the witness, said deposition to be submitted to the witness for reading and signing.

I further certify that I am neither attorney or counsel for, nor related to or employed by, any of the parties to the action in which this deposition is taken, and further that I am not a relative or employee of any attorney or counsel employed by the parties hereto or financially interested in the action.

IN WITNESS WHEREOF I have hereunto set my hand and affixed my notarial seal this 20th day of June, 1988.

Luke T. Lavin, R.P.R.
Notary Public, State of Ohio

My commission expires:

May 6, 1990.

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