IN THE UNITED STATES DISTRICT COURT FOR
THE NORTHERN DISTRICT OF ALABAMA
SOUTHERN DIVISION

ANTONIA TOLBERT, et al.,
Plaintiffs, CIVIL ACTION NO.
vs. CV-01-C-1407-S
MONSANTO COMPANY;
PHARMACIA, INC., and
SOLUTIA, INC.,
Defendants.
* * * * * *

DEPOSITION OF ROBERT G. KALEY, II
VOLUME I,

taken pursuant to notice and stipulation on behalf of the Plaintiffs Antonia Tolbert, et al., in the Law Offices of Lightfoot, Franklin & White, The Clark Building, 400 20th Street North, Birmingham, Alabama, before Angela Abbott Blankenship, Certified Shorthand Reporter and Notary Public in and for the State of Alabama at Large, on May 1st, 2003, commencing at 1:30 p.m.

APPEARANCES

FOR THE PLAINTIFF:
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FOR THE DEFENDANTS:
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STIPULATIONS

It is stipulated and agreed by and between counsel representing the parties that the deposition of ROBERT G. KALEY, II may be taken before Angela Abbott Blankenship, Certified Shorthand Reporter and Notary Public in and for the State of Alabama at Large, without the formality of a commission; and all formality with respect to other

procedural requirements is waived; that objections to questions, other than objections as to the form of the question need not be made at this time, but may be reserved for a ruling at such time as the deposition may be offered in evidence or used for any other purpose by either party as provided by the Federal Rules of Civil Procedure.

It is further stipulated and agreed by and between the parties hereto and the witness, that the signature of the witness to this deposition is hereby not waived.

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ROBERT G. KALEY, II, of lawful age, having first been duly sworn, testified as follows:

THE COURT REPORTER: Will this be usual stipulations?

MR. KELLY: Yes.

MR. KALEY: I want to read and sign.

MR. RODEN: He wants to read and sign, but other than that, yes.

EXAMINATION

BY MR. RODEN:

EXAMINATION

BY MR. RODEN:

Q. Dr. Kaley, I guess, just for the record, your name?

A. Robert George Kaley, II.

Q. And Dr. Kaley, your position with Solutia is?

A. Director of Environmental Affairs.

Q. And you have held that since what year?

A. Well, I have held the job at Solutia since the creation of Solutia in September of 1997. I had a similar position in Monsanto since, I believe, about 1994 or '95.

Q. Okay. And where are you physically located?

A. In St. Louis.

Q. Have you been in St. Louis since the inception of '94 of Monsanto?

A. I was with Monsanto long before '94. I have been with Monsanto in St. Louis since December of 1973 actually.

Q. Okay. In St. Louis?

A. Yes.
Q. Have you ever worked in any other facility or been anywhere other than St. Louis?
A. Not located there for any length of time.
Q. Your office, in other words?
A. Correct. Exactly.
Q. And who do you report to?
A. I directly report to Tom Bistline, B-i-s-t-l-i-n-e.
Q. What's his position?
A. He's an attorney.
Q. Is he general counsel or in-house counsel?
A. He's assistant general counsel, I believe.
Q. And who would work under you? What titles are people who work under you?
A. I have -- as Monsanto employees there's a manager of -- I don't know whether he's -- I think he's manager of environmental technical support and then

I have a person who's known as, I think she calls herself a literature specialist.

Q. What does she do?

A. She manages the literature that we maintain on PCBs and other issues.

Q. Like the various articles concerning health studies, etcetera?

A. Generally, yeah, a variety of things.

Q. Now, the environmental manager of technical support, you said worked for Monsanto, is that --

A. I'm sorry. After thirty years, I use them interchangeably. No, he works for Solutia. He came to Solutia in '97 just like I did.

Q. Does anyone working under you or above you work with or for Monsanto?

A. No.

Q. Does anyone above you or below you work for Pharmacia?

A. No.

Q. What exactly, and I know I have had the benefit of your depositions and transcripts, but tell me, since, in the last two years, what exactly do you do for Solutia.

A. Well, my primary responsibility is to remain knowledgeable to serve as a resource for issues around chemicals that were formerly made by, primarily, Monsanto, so primarily to remain knowledgeable as a company support of what we call Legacy Chemical, Legacy issues.

Q. What is the purpose for Solutia maintaining a support on Legacy products?

A. Well, there are a variety of issues that I have to deal with. There are numbers of regulations on the kinds of products that I share compliance within the company for. There are developments in the science that we need to be knowledgeable about.

Obviously, there are issues around litigation that I provide support to the litigation staff on science and technology issues. I provide science inputs on remediation issues associated with those chemicals.

We also have -- we still, because we were the primary manufacturer of polychlorinated biphenyls in the United States, we still get calls from former customers from outside the company, and I handle those calls.

Q. Now, who do you deal directly with at the Anniston Solutia facility?

A. Primarily Craig Branchfield.

Q. How do you spell that?

A. Branchfield?

Q. Yes.

Q. B-r-a-n-c-h-f-i-e-l-d.

Q. What's his position?

A. I believe he's manager of remedial projects.

Q. And do you frequent the Solutia

facility in Anniston?
A. I don't know what "frequent" means. Sometimes more, sometimes less. I'm probably there on an average of once a month.
Q. What exactly is the facility manufacturing today?
A. It manufactures a chemical called biphenyl which is a component of heat transfer fluids and it manufactures a chemical called para-nitrophenol p-h-e-n-o-l, which is a raw material for analgesics.
Q. Prior to that, when was it exactly that Solutia or, I guess, really the Monsanto facility ceased producing PCBs in the Anniston plant?
A. 1971.
Q. Now, do you, as director of environmental affairs, keep up with the various samples, the soil samples that are being conducted by various groups in the Anniston area?

A. From a very high level viewpoint, yes.
Q. What do you mean?
A. Well, I mean I see the results and I think about what they look like and, you know, overall and what they're telling us, but I don't necessarily look at each sample individually and try to determine whether it makes sense with the one next to it or things like that, but I have a general overview, overknowledge of the soil sampling results.
Q. Would that include Dr. Bonner's soil sampling?
A. I am aware of those. I haven't reviewed those in any great detail.
Q. Have you reviewed those in relation to the Tord Case?
A. Not specifically.
Q. You have reviewed soil samples in relation to the proposed settlement agreement between EPA and Solutia?
A. Yes. Again, at a very high level,

yes. I mean, I'm aware of, generally, the types of samples that have been taken, where they have been taken, the general levels, a general overview of the distribution of those levels.

Q. Can you tell me, with respect to that agreement -- I understand the, I guess the target level is -- is it ten parts per million? Is that the target level for remediation?

A. Well, okay, the current consent order under which we are operating has what they call a removal level of ten parts per million with a "M", so properties which have PCBs in excess of ten parts per million determined by a five point composite sample are subject to removal by Solutia under that consent order.

Q. And a five point composite sample meaning taken in five different areas?

A. Yes. Basically, they divide the area to be sampled into quarters and they

take a sample in the middle of each quarter and at the conjunction of those four quarters in the middle and add those together and analyze that as a single sample.

Q. What is the current situation with that sampling?

A. With the sampling, I believe we have sampled two to three of the areas, main areas that we have agreed to sample under the consent order. In that sampling, we have identified something like twenty-five or twenty-eight properties that had PCBs above the ten parts per million level and we have cleaned up, I believe, twelve or thirteen of those properties.

Q. Tell me about the area, and I know I have seen it, but I'm not sure I understand the areas that y'all have agreed to that there has been an agreement to sample.

A. Well, basically, when the

discussions with the EPA were going on, they had taken a map of Anniston, and somehow, and I don't know their logic, divided it into areas. I think they were numbered one, two, three, four, five, six. One of them is labeled "F" as in Frank, and whatever their rationale was for those particular divisions, we had to sample those in some priority, the ones closest to the plant first, the ones closest to drainage pathways, and then moving, as we moved farther away from the plant, those had lower priority.

Q. Is there a dimension involved in that; that is, an area of dimension? Is there a one-mile radius, two-mile or five-mile or ten-mile?

A. No.

Q. Is there any radius involved at all?

A. No, I don't believe so, not that I'm aware of anyway.

Q. Who decided the areas, the EPA?

A. Yes.
Q. Did y'all have any input into that?
A. Not really, no.
Q. Do the areas include properties outside what is called the flood plain?
A. Yes.
Q. Is it any particular distance from the flood plain?
A. Well, the distance is varied depending on -- I mean it's not -- the areas are not set by okay, here's the flood plain and you have to go half a mile past that or anything like that. I really don't know what their -- there's nothing, no rhyme or reason that -- I have never looked for one, but I don't see one obviously.

In fact, part of our strategy, our sampling strategy is to sample in a given area, sample the areas in and close to the flood plain first, moving away from that to see if there's PCB levels and if we get to a point where we're no

longer seeing PCB levels on a statistical basis, we may not have to continue to sample the rest of that area. We may move to another one of those areas where there are more likely to be PCBs found, if that makes any sense.

Q. Yes, it does. But that would be if the EPA agreed with that?

A. Absolutely, yes. We have to provide to the EPA our statistical analysis to try to justify such a decision and then they have the final decision and they have every right to say no, keep sampling.

Q. What I'm asking, Doctor, is this: assume that there are people that I represent outside the flood plain.

A. Yes.

Q. How can I assure them that they are going to be quote "tested" for PCBs, and if it exceeds the removal level, that it will be cleaned up? And that's why I'm asking the question, and I'm not sure

anybody can answer that question.
A. Well, I'm not sure they can either. I mean the first step, obviously, would be to -- there is a map that exists.
Q. And I may have it somewhere.
A. I'm sure you do, because if you got a copy of the consent order, I'm sure you do have it, that lays outs those particular areas. The first step would be to determine where those people lived in priority -- within those areas.

If they live within those areas, then they are certainly, more likely than not, to be sampled. If they are outside of those areas, under the current consent order under which we're operating, I think there's less likelihood that they would be sampled.
Q. Okay. Do you know whether or not the agreement to, or the proposal for those areas was determined by any factors that were used to determine that? Do you have any knowledge of that?

A. Okay, now, let me try to clarify something. You keep talking about a proposal. I'm talking about the consent order under which we are currently operating.
Q. Okay.
A. Now, there is a proposed consent decree which is a different animal, so I'm talking, and I hope you are, about the consent order under which we are operating. And my understanding, as I said, I don't really know how that was conceived. But my understanding is it was based on sampling that the EPA had done in the Anniston area and roughly encompassed those areas where, in their sampling, they found some properties that had PCBs on them.
Q. Now, what is the difference between the order in which you are working and the consent proposed as far as the area is concerned that would be sampled?
A. Well, as far as I know, and I'm not

the expert on the details. My understanding is primarily with regard to residential sampling. I believe the areas are roughly the same. The difference would be on an established cleanup level and which properties and the number of properties that would be required to be cleaned up.

Q. What would the established cleanup level under the proposal --

A. Under what they call a streamlined risk assessment that EPA has carried out, the cleanup level would be one part per million. Any property with a composite sample above one part per million would be required to be cleaned.

Q. But the areas would still be the same?

A. That's my thought as I sit here. I don't know that for sure. Now, part of the proposed consent decree is additional investigations leading away from our plant, and that's certainly areas to be

identified that were impacted by PCBs. Those would be sampled.

Also, the consent decree, the current consent orders address only residential properties. The consent decree would address other kinds of properties; public access properties, things like that.

Q. Schools?

A. Yes. Basically, any property in the area.

Q. And, again, in the areas proposed in the sampling?

A. Yes, I believe that's true, although I'm not -- please, don't hold me to exactly that, but it's basically that general area.

Q. Tell me this: who was involved or is involved, I should say, with working with the EPA to come up with the proposal?

A. Primarily Craig Branchfield from Solutia. I have been involved in some of the discussions, but he's the man on the

ground. He's the one that understands what -- I mean, the consent decree talks about the Anniston PCB site under terms that are EPA's terms for defining a site, so I don't know that it's exactly the same as a consent order, but I believe it's generally the same.

Q. Okay. Can you tell me why the removal level was changed from ten parts percent million to one part per million?

A. Well, the emergency removal action or level was primarily determined by EPA by reasons I don't know to believe that was the level that needed to be cleaned up immediately. The one part per million, as I said, is a more robust number, I'll use that term, determined by the streamline risk assessment that was done by EPA, so it's more a risk-based cleanup level.

Q. What do you mean by streamline risk assessment?

A. Well, it was primarily done without

a lot of it -- it was based on the data that the EPA had available to them at this point rather than requiring a lot of additional sampling to determine level, so that's primarily the streamlined nature of it.

Q. When you say risk assessment, what exactly do you mean?

A. Well, a risk assessment is, in EPA's mind and the contractors that do them for EPA and the contractors that do them for us is a very formalized process under which, basically, it's a comparison of sampling results to numbers that the EPA or someone has generated to address the theoretical risk associated with exposure to those levels.

Q. Are there risks associated with exposure to certain levels of PCBs?

A. Well, in the view of risk assessment, there is, by definition, because what the EPA, primarily, and what they have done in this case is take

results from animal studies and, primarily, these are based on results testing whether PCBs cause cancer in animals, and this is for any chemicals, not just PCBs, and determine the concentration in which that happens in animals, apply a bunch of safety factors to account for potential differences between animals and humans, potential differences within animals, etcetera, and then come with a number which, in their view, is protective of human health, so they come up with this, in this particular case it's called a cancer slope factor. They apply that number to a concentration to determine, in their view, whether there is some theoretical risk. The true risk, and EPA acknowledges this, could be zero in the risk in the way you and I think about it.

Q. Right.

A. But their theoretical risk is what

determines these cleanup levels.
Q. Well, obviously, the issues here are, in fact, PCBs carcinogens. Do they cause cancer?
A. Yes.
Q. And I think there's a big disagreement with that. I don't think you and I would agree with that.
A. I believe we would disagree in humans. I don't think we would disagree in animals, but I think we would disagree whether PCBs cause cancer in humans, yes.
Q. And I assume you are familiar with the various clinical studies, if you will, that have tried to relate PCBs with cancer and other problems?
A. Yes, I am.
Q. What is it that can Solutia does agree, if they would, that PCBs causes at certain levels?
A. We believe that PCBs, at high levels of exposure in humans, are associated with dermal skin effects, possibly

including something called chloracne. I personally don't necessarily agree with that, but that's, you know, the company has acknowledged that that's a possibility. And then also with transient elevations in some liver enzymes, those have been reported fairly consistently in some studies.

Q. And do you have any understanding, I guess, of what the transient elevations in liver enzymes, what risk factors that would cause?

A. My understanding is probably nothing specifically. I mean, when they have been reported, they seem to go away if the exposure is reduced or eliminated, and in those populations, you don't see any evidence of further progression to frank disease.

Q. Is there any difference in risk exposure depending on the chlorination of the PCBs?

A. Well, again, I think if you look at

animal studies, there seems to be, and certainly, if you look at cancer studies specifically where most of the study has been focused, there does seem to be an appearance that the more highly chlorinated PCB mixtures are more potent carcinogens in animals than the lower chlorinated mixtures. But, again, we don't see health effects in humans particularly, so that we can't really make that kind of assessment on the human studies.

Q. What is the -- what happens when you heat up the PCBs as far as the chlorination of the PCBs? Does it do anything to change the particular chlorination of them?

A. Well, if -- I guess there's two ways to answer that. One is if you look at an individual PCB molecule, heating it will not change the amount of chlorine on that molecule by itself. I mean, if you're in a reaction vat and you have chlorine in

there and something to promote the reaction, then, obviously, that's how you make PCBs, so you will continue to add, but if you have got a product sitting here on the table and started heating it up, then there's nothing on an individual molecule that's going to change, as I said, the numbers of chlorines on that individual molecule.

If you look at the mixture as a whole, if you heat a vat or a jar of PCBs and start raising the temperature, then the more lower chlorinated materials may very well evaporate into the atmosphere and so the mixture as a whole may be shifted to higher chlorination, but that's only because you're, by heating them, you're removing them to more volatile materials.

Q. You're transferring them?

A. Right, you're taking them out of the mixture here in the vat and you're putting them in the atmosphere so that by

-- you know, that leaves the more highly chlorinates ones in the vat, but it doesn't change the chemistry of an individual PCB molecule by simply adding heat, and that's why they're good. That's why they were used in many of the applications they were used is because they are stable under conditions of heating.

Q. Well, as I understand, just from my limited knowledge, that the PCBs that were manufactured at the Anniston plant, the highest number of, as I understand, the highest number chlorinated was like seven or eight or was it even that high?

A. Well, I mean, I know you don't want a chemistry lesson or a PCB manufacturing lesson, but all the PCBs were very complex materials, very complex mixtures of materials, and as you go through the product sequence from 1221 to 1242 up to 1260 or 1268, then the number of chlorines on the molecules in those

mixtures tended to increase so by the time you got up to 1268, certainly, there were individual PCB molecules with nine or up to ten chlorines on those rings in that product.

Q. Okay.

A. But now, there was no product that was all what we call decachlorobiphenyl, the highest biphenyl with ten chlorines on a ring. We actually considered making that and some people did make that as a product, but that was a white solid as opposed to many of the other materials. That was a pure single compound, but that material itself was a component of some of the other mixtures and, most notably, would have been a component of Aroclor 1268.

Q. What was the majority of the PCBs manufactured in Anniston as far as the number of chlorines?

A. Well, that's a difficult question. We really don't know the answer to that

question because we didn't manufacture PCBs by the number of chlorines on the ring. We manufacture PCBs by these complex mixtures with average chlorination.

Now, my understanding is that, by far, the most -- the product of most volume in Anniston was Aroclor 1242 and that product is primarily biphenyl molecules with three and four chlorines on ring so by extrapolation, you know, pushed to an answer, I would say probably three, four, maybe five chlorines on a ring was the most, but there would have been a distribution including all the possible levels of chlorination.

Q. What about the still bottoms or the by-product of the manufacturer of PCBs, did they have any more chlorine -- number of chlorines in them than the mixture itself?

A. Yes, I would think, in most cases, that would be a safe assumption because

of what distillation is. Distillation a process by where you basically take a mixture to still off or remove the lighter materials in that mixture. In this case, it would be the less chlorinated PCBs leaving the more highly chlorinated PCBs in that still bottom along with a variety of other materials, but I would expect from any given process, that the still bottoms from the distillation, you know, the products from the distillation of Aroclor 1250. For example, the still bottoms would have a higher level of chlorination than 1254 itself by some unknown amount.

Q. When we talk about still bottoms, and I don't know the process, but are we talking about a large vat of some sort that these mixtures are being made?

A. Primarily, yes.

Q. How large a vat?

A. You know, I don't really know the answer to that. I've never gone in and

looked at the size of the vats. But it's primarily a large reaction vessel.

Q. And as far as still bottoms, can you quantitate that for me as far as how much would be in a vat?

A. Again, I don't know.

Q. Do you know what was done with the still bottoms?

A. Primarily, once that vessel -- the reaction that completed, my understanding is that they were drummed and put in the landfill at the site.

Q. Were there any of them washed into the sewer system?

A. Not that I know of.

Q. Or drainage system?

A. Not that I'm aware of.

Q. Does Solutia --

A. I mean they wouldn't be washable. They're very viscous, hard, solid. They're really not the kind of thing you can move with a hose or something like that. It wouldn't make any sense.

Q. Kind of like burning something on the stove and trying to scrape it out?
A. Yes, exactly. It's basically the consistency of road tar, I mean very much the same.
Q. Does Solutia admit or agree that there have been PCBs that have escaped from the Solutia facility?
A. Yes, I think we would agree that there has obviously been, in drainage pathways, some PCBs have moved from our facility to off-site, yes.
Q. Is there any agreement as to the quantitative amount?
A. I don't believe that's determinable. I don't know who I would be agreeing or disagreeing with. I've never heard an amount, so, you know, I don't know.
Q. Well, you probably, like me, have seen documents that talk about several pounds per day and that type thing?
A. Right.

Q. Is that something that we can agree on or is that something that's still in dispute?
A. Well, I think we can agree that those documents exist and the numbers are there. I think, knowing the state of analytical chemistry at the time, there is some uncertainty around those numbers, but I think they certainly stand for the proposition that PCBs were in those waste waters.
Q. Okay. When you say there's a certain -- the analytical chemistry was not available at the time, what do you mean?
A. Well, I'm just talking about the uncertainties in the measurement whether, you know, if you're getting -- measure one hundred parts per million, is that because that sample really has one hundred parts per million or the sample before it had one thousand parts per million and it carried over into that

sample or a number of things.

It is just like everything else that we deal with on a daily basis, the technology and abilities to do analyses with PCBs have gotten much better over the past three decades or four decades that we have been using, so there's just a lot of uncertainty around those numbers.

Q. Do we know or does Solutia know to the point where they could admit how those, whatever the number is, PCBs escaped or got out of the facility?

A. I think we have -- I mean starting in 1968, we began to look at that very question; are they in there and how are they getting out, and I think we have some understanding of how some of them got out at least.

Q. When did Monsanto and Solutia start trying to determine the number or how much was escaping into the environment?

A. Well, I believe we started -- I

think you probably have been through the history, but the report of PCBs in the environment really began back in late 1966 by some Swedish researchers and, you know, there was a learning curve that Monsanto and others had to get on and climb to and it took us a couple of years to get on that learning curve and develop the analytical techniques, but I believe, if I've got my numbers right, by '68, we were actually taking samples in Anniston and by '69, you know, waste samples and water samples and by '69, we were actually able to start making some of those kinds of measurements that are reported on the documents that you're referring to.

Q. Where were they coming from? How were they getting out? What means --

A. I think, primarily, there were several ways. One was -- I think there was a waste stream from that process that was primarily -- hydrochloric acid is

generated when PCBs are manufactured in a gaseous state, then it's bubbled through water to make it liquid hydrochloric acid and some of that was discharged as a waste through the plant through what I think are called the limestone beds or limestone pits where the acid was neutralized and there's some PCB carryover in that.

Q. Right.

A. There's certainly reports, you know, if they were spilling in the department that a hose or washing would be done with detergent and that would mobilize the PCBs into basically the storm water drains at the plant. It really wasn't a waste stream as much as it was storm water and process water.

Q. So waste stream and storm water, you're saying that's synonymous, are you not?

A. Well, no, they're not synonymous, but there wasn't -- it wasn't a waste in

the terms of a chemical or organic waste that you think about coming out of a process. I mean it was primarily water and it was either this hydrochloric acid coming from the scrubber's process or it was storm water moving things along or cleanup water, but those kinds of waters those, what they call noncontact waters, and the storm waters were mixed in the sewers at Anniston so all that was going out at the same point.

Q. Is there any agreement, I guess, with Solutia that any of the landfills had escapes or exits of the PCBs from them?

A. Well, when we became aware of the situation in '93 and started measuring the trace levels of PCBs in the storm waters, yeah, the run-off waters from those landfills finding, you know, a part per billion or a few parts per billion, I mean, those kinds of levels were being carried away by soil erosion or soil

pickup from the landfill, so to the extent that those trace levels were being released, I think that we certainly agree there. I see no evidence that there were any large quantities of release from those landfills at any point in time.

Q. Even prior to '93?

A. Even prior to '93, right. I think, you know, as -- when we closed the process and basically sealed those, the cells where PCBs had been disposed of, largely, I believe that probably shut off most, if not all, other than these trace levels of discharges from the landfills.

Q. You have two landfills; correct?

A. That is correct.

Q. And each landfill has a number of cells?

A. Well, certainly, the south landfill has a number of cells. I'm not so sure about west. It may have been a single cell landfill. I don't know as much about that.

Q. And you're not sure?
A. I mean, there's clearly a west landfill and it had at least one cell, but I don't know that there were a number like there were in the south landfill.
Q. And when you say you basically seal those, and that was in what, the seventies?
A. Well, the PCB landfill was generally closed in early 1972, 1973, but as we got into what was called the RCRA, the R-C-R-A process, the Resource Recovery and Conservation Act then we had to take additional steps on all of the landfills, as did all chemical manufacturers, to be sure that they were brought up to compliance, so in the late 1980s, additional work was done on closing those landfills, so my confidence increases as we move through those various stages. But certainly, by the time of the RCRA closure in '98, I believe there was, other than just the little bit of soil

runoff, probably no significant contribution to PCBs off the plant site or off the landfills, certainly.

Q. But is there anything in the documentation that measured the numbers of PCBs escaping from either of those landfills prior to '89 or prior to, you said '93 earlier, but any time in the eighties?

A. Well, we always had a storm water permit when the storm water permit went in so I think we have some, probably not extensive, but some sparse data on storm water. We were also beginning in the early-1980s and continuing monitoring groundwater, water going out from under the landfills, and, again, there are data through that early-1980s, well mid- to late-1980s time frame that indicates that PCBs were not leaving by the groundwater route either, so there are some, they are not extensive, but there are some data that stand for those propositions.

Q. When was it that you had to get the storm water permits? When would that have been?
A. I'm sorry, I don't know the answer. I don't know when the MPDS process really went in. I think it was the mid-1970s, I think, but don't hold me to that.
Q. Okay. In reading your testimony, Dr. Kaley, in the Abernathy case, you made mention when asked the question about human health studies that you did not feel a study is necessary nor appropriate based on the understanding of the size of the population and you go on to say that to do an appropriate epidemiology study, it takes thousands of people and you don't believe that there are thousands of people exposed in the Anniston community. Does that refresh your memory about it?
A. I believe that's a fairly -- I mean, I remember that question and I'm sure that's what I said, if you have that

written down, and I think that's probably true.

Q. And I believe you further say that a lot of this was based on the blood levels you had seen?

A. Yes.

Q. And I guess, in that particular course, was the number of those plaintiffs in the Abernathy case which, I believe, is like thirty-five hundred or so.

A. Yes.

Q. Have you seen other blood levels other than those?

A. Yes.

Q. What blood levels have you seen?

A. Well, seen generally -- I mean, again, I haven't spent a lot of time on the details, but I've seen the blood level data from the Tolbert litigation.

Q. Does that change your mind, in any way, with respect to a need to have a health study or what they call, I guess

in this testimony, a human health study?
A. No. I think the distributions are generally -- I mean, you see, you know, a large proportion, a majority of the people are nondetects or certainly right at the level of detection. You see another group of people who are what would be called normal background levels throughout the United States, and then there is a smaller group of people that do have current or past exposure based on the blood levels.

But again, compared to you really need to have a epidemiological study to look for whether exposure to PCBs is associated with causes of death, it really isn't -- you know, I guess it's really what you're talking about. If you're talking about an epidemiology study or mortality study, you know, I mean, you have to have large numbers of people who have died that birth certificates can be obtained from and I

think that would be very difficult in the Anniston area to do that kind of study. If you were just doing a survey of what levels people have and whether they have complaints, that's a whole different study and those are fraught with difficulties because all of us have health effects and we can attribute them to a variety of different things.

That's why mortality studies are typically are done to determine those associations because they are kind of hard and fast. You had a person, that person died of something, and you know what it was, and, you know, the difficulty there is you don't always know what the exposure was, so it's just going to be very difficult to do anything that's going to be meaningful in that population.

Q. You're differentiating a mortality epidemiology study with a large health study?

A. Right, what I would call a morbidity study, right.
Q. And you're saying that a mortality study is more appropriate than a morbidity study?
A. Well, I think it's more appropriate to a make a final determination on whether, for example, PCBs are associated with human cancer or other major diseases.
Q. What is the level that you would consider quote "nondetect" unquote or what is the level that is considered nondetect?
A. Well, that depends on -- I don't know. Without being flip, that depends on the laboratory and what their capabilities are. I mean, I think most of the detections in the samples I have seen from, you know, whatever litigation have been, you know, three parts per billion in blood and five parts per billion in blood, but certainly, we know

from literature and where you take larger blood samples and do a lot more work-up, I mean, you can measure clearly in the parts per billion and parts per trillion in blood, so your question is really an analytical question if that's what you really meant.

Q. Well, I guess what I'm trying to determine is what does Solutia consider as being nondetect as far as parts per billion in the blood?

A. Well, again, without being flip, it's whatever the laboratory that did the analysis reports as nondetect. I mean, we don't have an opinion on what that number is. It's what the laboratory can or can't do.

Q. What is the normal background levels?

A. Okay. That's a different question.

Q. Right. All right.

A. As I sit here today, I would say that number one, you have to look at --

there's two issues; what is the average background level and what is the range of people not otherwise exposed to PCBs that still have PCBs measure inside their blood. I think the average is probably in the maybe three-to-six-part per billion range, so if you looked at all the studies of quote "unexposed people" and averaged all their values, you would see, you know, an average of three to six parts per billion. That average, though, is made up of people who have lower levels and higher levels.

Q. All right.

A. Okay. Those higher levels, I think, you know, I mean, the published data say those higher levels go up to maybe twenty parts per billion at the ninety-five percent level. In other words, ninety-five percent of the population has levels twenty parts per billion or below.

I think, you know, the ATSDR is trying to make a case for it, and I think

there's probably some slight justification that that number may be somewhat lower now, but we don't have the data and we don't know the answer to that, so as I sit here today, I would say that people with up to twenty parts per billion would be considered within the background range.

Q. And obviously, there's a difference of opinion about that or is there?

A. I don't know whether there is or not, no. I mean, I think there are people out there who would disagree with me, but as far as being able to document that on either good scientific results or what studies have been done, I don't think there's anything that really -- I mean, if the literature said different than what I say, I would go with what the science says, but I just don't think it's out there now.

Q. The ATSDR's report, it mentions a figure in there, and I may be able to

find it -- well, the tox profile; are you familiar with the tox profile?

A. Yes.

Q. They talk about mean serum level?

A. And that's what I was calling average.

Q. Is that the average?

A. Yes, mean and average meaning the same thing.

Q. So average, when you say average background being three to six parts per billion, but then when you look at the total number, you're saying that the higher end of that is twenty?

A. Up to twenty, yes.

Q. Okay. And does that -- do those differentiate between those who have been exposed and those who haven't been exposed?

A. Well, all of us are exposed. I mean -- and some of us without occupational exposure or without any other identifiable source of exposure that you

or I could point to and say oh, there's where I got my PCBs, that range goes up to twenty and maybe it's because a few more fish, maybe it's because we eat more beef, you know, and for whatever reason, the beef we ate may have lower or higher levels than someone else, so there's a lot of reasons that could explain that variation in the human population. You know, maybe my body is better at getting rid of them than your body. Maybe I'm older. If I'm older, my levels are going to be higher because, you know, one of the properties, unfortunately, of PCBs is they are not easily eliminated, or some of them aren't, from the human body, so as you get older, that continuing input of PCBs tends to build up in your body, and that's well documented.

So there are a lot of reasons to explain that range, but I guess what I'm saying and trying to communicate is that if we are going use that magic

twenty number, you can have nineteen or twenty and still not be quote "exposed" and you're still in normal background. You don't have to go around and say okay, I've got more in my blood than the next guy, how did I get them, or where did they come from because it's all within that normal range.

Q. Well, when the Tox Profile says that the mean PCB serum levels range from point nine to one point five parts per billion in recent years in individuals who did not have diets high in fish from waters contaminated with PCBs; is that what you're telling me?

A. Yeah. I'm using -- I think there's two qualifications. One is they're qualifying recent years, and you think with all the attention on PCBs, you think there would be tons of data. There are not tons of data. There are a few studies here and there. For the purpose of this statement, and I don't know

exactly how many they looked at. We could go find out. I think they looked at two or three very recent studies and there's no doubt the levels in the environment are going down, okay.

Q. Right.

A. So my number, my three to six is, what I'm saying, is the same as their point nine to one point five. I don't think there's a lot of difference in their -- a factor or two is not a big difference in my mind, and I'm looking at a broader view. I'm not looking at the two most recent studies. I'm saying over the last decade or last fifteen years, if you look at all those studies, so I don't disagree with this, nor do I think it's very different from what I'm saying, but that's, again, that's the average. That's the mean. That's my -- their one point five is my three number. They are still made up with people that could go up to tens or twenties that help make up

that average.
Q. And there are people, then, you say, that have twenty parts per billion in the general population?
A. Certainly, to the data we have available to us, that still seems to be a reasonable number.
Q. And that might be because they were more highly exposed than those who have one point five?
A. And through their normal living without any unique source of that exposure, yes.
Q. Would you agree that a person who has currently three parts per billion had more, assuming he was living, had more fifteen years ago than he has now?
A. No, I wouldn't. I absolutely disagree with that.
Q. Why is that?
A. Why should they? I mean, if I've got three now, I would say fifteen years ago, my number was lower because, as I

explained, we're continuously exposed to these low levels of PCBs and as we get older, our number tends to increase.

Q. So you don't agree that you can take a person now that has five parts per billion and extrapolate fifteen years back and determine how much he had in his blood serum fifteen years ago?

A. I absolutely say you cannot do that. A person within in the normal background range, you can't do that with, absolutely not, because you have to correct for that background that you have and I have and Mike has and everybody has. You have to correct for that background.

Q. Is that true for people in Anniston?

A. It's true for anybody that has PCB levels in the background range, yes.

Q. And those that you have seen in Anniston with high levels and when I say high levels, say fifteen or twenty parts per billion. You may not consider that high, but let's assume that to be the

number.
A. Okay.
Q. Do you agree that those people are at a higher risk for a disease or problem than one in Anniston that has only one point five parts per billion?
A. The answer to that is no for a number of reasons.
Q. Tell me those reasons.
A. Number one, I think the reason is that, I mean, we already talked about a potential disagreement, but certainly, the view that I hold that PCBs are not associated with long-term serious health affects with the potential exceptions of dermal effects and transient liver effects. So I don't necessarily believe -- I don't believe, at whatever level your PCB level is, that you are at real risk, not theoretical, not regulatory risk, at real risk of developing disease as a result of those blood levels, okay, so that's number one.

And then going, you know, even further back than that, all of those levels are very low background levels whether it's one or fifteen or twenty and I just don't believe there's any risk associated that.

Q. They are low background levels within --

A. I mean, they are levels within the background range.

Q. The general population?

A. Yes.

Q. You don't mean -- when you say background levels, you don't mean background levels as opposed to like fifteen years ago, we're not using that?

A. Well, I don't think it matters.

Q. Okay.

A. I think people that had fifteen or twenty parts per billion fifteen years ago where it was more likely that they were in the normal background range, I don't think they were at any more risk

than the people that have one or two or three now because I don't believe there's any risk associated those kinds of blood levels.

Q. Is there a level that you would consider a risk to a human to develop a disease, if you will, from exposure from the levels that they have in their blood?

A. Okay, I don't -- based on my understanding of the human literature where we have seen levels up to, you know, twenty-five hundred and three thousands parts per million in some of these workers, twenty-five hundred parts per billion or two to three parts per million in blood, we have not seen, other than skin effects, health effects in those workers, so based on the range of human populations that we have been able to study and that have been exposed to PCBs, I do not believe there's risk of human adverse health effects.

Q. At all?

A. Other than the liver and the skin, so I can't pick a number. Now, if you go into the literature, there are some papers that will try to pick a number. I think an early paper said you had to have at least two hundred parts per billion to be at risk of chloracne.

Now, I'm not sure that that paper really stood for that proposition, but I mean you could go out there and find those kinds of things. But I think, in general, looking over the whole literature and looking at the blood levels of people who have been studied, I don't think you -- it's not that I think -- you don't see those risks. You don't see those diseases, let alone risks.

Q. Of course, you're saying you don't see those in certain studies you're relying on?

A. Yes. I mean, if you're going to, you know, I mean, the proposition is if you want to know what happens to people

when they are exposed to a chemical, the first thing you do is go look and see if people somewhere have been exposed to high levels of that chemical, for instance, people that work with it every day, and if they're not getting ill or dying from those exposures, then there's no reason to believe people who are exposed at low levels down to zero levels are going to be ill or die from those exposures.

Q. And of course, you're saying the people that worked around it were more highly exposed than someone out in the community; that's your belief?

A. That's a general proposition, I would say, yes.

Q. And were those people that were exposed working around it, were they protected in any way? Was there any protective equipment?

A. You know, in the early days, probably not. In later days, I think --

I mean, I certainly know our workers had, you know, work clothes that they could take on and off. I think they were told to wear gloves if they were in direct contact, but I think we know from talking to people that they didn't, that they were contacting those materials.

I mean, you hear the apocryphal stories of people using PCBs as grease removers and things like that, so I mean, there was normal hygiene protective equipment available to people. I'm sure some wore them and some didn't, but there were no extraordinary measures that you would see like in a plant where you would have to wear a respirator because you were working with some vaporized material or something.

Q. Would one be at a higher risk for exposure to PCBs if they consumed it as opposed to inhaled it?

A. No. With PCBs, my understanding of the studies is that it's, you know, if

it's getting in, it's getting in.
Q. Right.
A. Whether it's inhalation or dermal or ingestion, however it's getting in that those are all ways that PCBs can get in your body and there's no one that's really different than any other, I don't think.
Q. I take it you are familiar with the various studies, Brown, Bertazzi, Sinks?
A. Yes.
Q. And I'm sure you have read those?
A. Yes, I have.
Q. And do you agree or not that those studies do indicate an association of exposure to PCBs and cancer, for instance?
A. Okay. I believe that some of those studies report associations between purported or real exposure to PCBs and some individual cancers on an individual study basis, but if you look at all those studies taken as a whole, you do not see

a consistent picture of associations with PCBs with cancer at all or any specific cancer.

Q. And when you say lack of consistency, you mean a lack of consistency with respect to the particular site of the cancer?

A. Or all cancers combined, yes.

Q. In other words, one may have gastrointestinal colon cancer and one may have some other, and I'm not sure --

A. Well, they're both gastrointestinal. It's never been colon.

Q. Gastrointestinal, and one was some other cancer?

A. That's correct. You see one -- this here, this there, but, you know, I mean, you expect that. The epidemiologist, and I'm not -- the epidemiologist will tell you that, you know, one out of every twenty things you look at is going to be positive just by chance. That's why you

can't rely on a single study. You have to go look at these studies where, arguably, all those chance occurrences are going to average out and if there's something really there, it's going to be there in a preponderance of the studies.

Q. There are epidemiologists and toxicologists, as I understand, that do take those studies and state emphatically that they prove that cancer is, in fact, related to PCB exposure. Would you agree with that?

A. Would I agree that there are people that say that?

Q. I mean you have read that, I'm sure?

A. I guess you would have to look at how carefully they're saying it. I think if you go to the literature where people are being, you know, it's written on a piece of paper and they are being judged by what they say and it's there for posterity to look and think about, I think they'll go to say that there are

suggestions or there are associations we just talked about.

In a given study, there are some that report an association with exposure to PCBs and some individual cancer. Yeah, people say that. I'll say that, but I don't know that you could go to the literature and really find someone that says I've done a good weight of the evidence review of the PCB cancer literature and I know PCBs cause "X". Now, when they say that -- they might say that.

Q. Well, you have seen them say that. You have read testimony to that effect, have you not?

A. Yeah.

Q. Okay.

A. Although, again, I'm going to qualify that because I think, again, even these guys that -- well, I'll be nice -- even these guys that are saying it are generally pretty careful about qualifying

it. They'll use the word "contributed" or something like that, you know. Even all that stuff I've read, I'm not sure I've ever seen anybody say PCBs caused this person's cancer period, only PCBs period.

Q. You've never seen a doctor say that?

A. I may have, but I can't sit here, oh, yeah, I remember that guy saying that.

Q. Now, is Solutia, and I think we talked about this earlier a little bit, but is Solutia going to, in fact, are they doing a risk assessment on the Anniston residents?

A. All right, under the terms of the consent decree proposed in federal court, there will be a human risk assessment done and an ecological risk assessment done and, obviously, human risk assessment will purport to analyze the risk to humans associated with various levels of exposure, and the ecological

will look at the risk to the environment. Solutia is going, under the terms of that agreement, would have the responsibility for the ecological assessment. The U.S. EPA would have the responsibility for the human risk assessment.

But the risk assessment is really an assessment of the potential risk to humans from the exposures that could happen to soils or whatever in Anniston. It's not -- neither one of those is really a risk assessment of people in Anniston.

Q. That's what I thought. I mean, because, obviously, the EPA is not, I don't think, geared to a quote "true human risk assessment"?

A. Well, I don't know what that means. I don't think -- nobody -- you know, with all due respect, I don't think that's a meaningful term because I don't think anybody does that.

Q. Well, for instance, CDC, would they

be more equipped?
A. Well, I guess I don't know what you mean really by --
Q. I'm not sure I know what it means either.
A. I know, and the risk assessments that are being done, without getting into a lot of detail, basically say here are the potential exposures, given those exposures and given these numbers that we can calculate that relate to possible or theoretic risk, I basically multiply my concentration in the environment by this potential risk number and come up with a number that says this population has so much risks and that risk is either acceptable or it needs to be reduced by doing something. You can do that for the human and you can do that for ecological. That's what risk assessment is all about.
Now, if you're saying by human risk assessment, I'm going to take this

group of people, I'm going to take all their blood levels, and I'm going to do some calculations and see if they are at more risk -- I don't know if anybody does that.

Q. Okay, well, that's what I'm asking. Is that something that would be considered or necessary to a true human risk assessment; that is, determine the blood level in the serum of those humans to determine what risk they are at?

A. Well, I mean, somebody could do that, whether it's official or making a call, you and I both know there are people out there that have done that in other litigation. They're going to do that in this litigation saying if that person has that much PCBs, therefore, they are at that much additional risk or they won't probably quantify it because they don't know how to quantify it, and that's the problem.

They'll say it's additional

risk, but they don't know how to quantify it because there are no -- you would have to have human data where you had known human exposures getting to known human illnesses to calculate the relationship between blood level or any other measure of exposure and illness and you don't -- I'm sorry, you don't have the end points to make that extrapolation so you can't really do the calculation.

You can say, because of what the literature says, there may be some theoretical risk, but in any of this, I have never seen anybody calculate what additional risk some person is at because, number one, all of these risks are population risks. They're not individual risks.

Q. What do you mean?

A. Well, any risk assessment, it's averaging procedure, okay, and it talks about whether all the, you know, all the beavers in the pond, you know, one in a

million might get some end point. It's not -- Bucky Beaver may or may not get sick because he's been exposed to PCBs.

You can't do that under the risk assessment process because it's a broad process looking at broad characterization of risk and it's looking at population risk. It has nothing to do with individual risk.

Q. Are you saying --

A. It can't, because there are so many things that drive individual risk. You can't pick that person out.

Q. There's other compounders that --

A. Or any number of -- yeah, you can call them that if you want to. Lifestyle factors, you know, genetics, all sorts of things, and this whole process is based on averages and generalities and extrapolations and safety factors that builds so much uncertainty into the process it can't be I driven down to an individual.

Q. Are you saying the risks that are quote "associated with PCBs" is theoretical?
A. In risk assessment, as applied it is a risk -- it's a real risk to an animal.
Q. Okay.
A. Or it is a real end point? It's not even a risk. You've got animals that are sick or are dying and this isn't just PCBs. You take that -- how much it caused that animal to get sick and divide it by ten and you divide it by ten again and you divide it by ten again to come to a factor where you're comfortable there's enough -- you have put in enough uncertainty factors in there to say okay, way down here, a human exposed to this level isn't going to get sick like this animal up here at these very high levels did. All this is uncertain in here.

There's no certainty that even a human exposed to this level would get sick. It's all been done on mathematics

and policy, frankly. It's policy. It's safety factors. The EPA says we're going to put in ten for species uncertainty. We going to put in ten for individual uncertainty. We're going to put in ten because we have a NOAEL instead of a LOAEL -- the other way around, a LOAEL instead of NOAEL, I'm sorry, NOAEL or LOAEL, all caps.

Q. Meaning?

A. No observable adverse effect level or lowest observed adverse effect level. Thank you, that's good for clarification.

And so it's policy on how we are going to protect people in risk assessment, and so when you're talking about risk assessment and all that stuff, it's uncertain and it's population-based. It's not individual.

Q. So why do you do a risk assessment?

A. To help the regulators and the regulated community come up with, in general, a cleanup level or a no-effect

level that, under the terms of all this safety, we can all agree if we get to that level, we're cool.

Q. But if there are no risks associated with exposure to PCBs to humans, why go to all that trouble?

A. Well, again, you're using risk one way and I'm probably, in the regulation, using it another way. You're, you know, scientifically, a layperson saying risk means danger.

Q. Correct.

A. Okay. Risk in these -- I mean, it starts off with the same definition but the danger, at one point, is real, but you have gotten so far away with all these uncertainties and safety factors that the risk down at this end becomes theoretical based on -- the risk to humans is theoretical based on the real risk when an animal is at a much higher level of exposure.

Do you come up with a number?

Yes. And that's how it's regulated. It says if the risk is one in a million to one in ten thousand, depending on the conditions, that's probably okay. The U.S. Government -- the EPA will accept one in a million to one in ten thousand risk to the population for a cleanup level. That's the way it is.

But that doesn't mean that if you've got a one in ten thousand risk level, that in a population of ten thousand people, one person is going to get that illness. One person might. Ten people might. Zero might. But that doesn't mean it came from the exposure you're protecting against. It's just a way for all of us to come to an agreement on what we need to do to clean up a situation.

Q. So the known factor, i.e., the exposure to an animal, a fish by a certain level of PCBs causes that animal to either die or be sick?

A. Right.
Q. What's that level?
A. It depends on the animal and it depends on the end point you're talking about. And I'm not going to be able to give you numbers, but it takes, for example, it takes a lot more to give a rat cancer than it does to cause an otter or a mink to have reproductive failure.
Q. Okay. And so you take that as being known?
A. And there are all sorts of numbers.
Q. You take that to be known and you're saying that you don't know the level, if there is a level, that causes that same problem to a human?
A. Yes, I'm saying that and I'm saying that we have never seen those problems in humans to know that level, but we will all agree that, because we see it in animals, we're going to apply all these safety factors and we're going to clean up to that level for humans. Or animals

may be involved.

I mean, the ecological risk assessment is doing the same thing for the little beasties and, you know, if there are minks in Choccolocco Creek, then the risk assessment will take into consideration the reproductive risks in minks at some level of PCBs and we'll have to protect against that. And that may be even lower than what it is for humans in Anniston and that might not make people happy but it could easily come out that way.

Q. Sure. But even though you're doing a quote "risk assessment", human risk assessment, you're not agreeing, as I understand it, that the exposure that the humans in Anniston have is not causing them any uncommon risk or unduly risk?

A. All right, I'm going to restate my answer. I'm going to answer what I think you meant to ask and maybe you did.

Q. Okay.

A. I do not believe that the exposures in a stone as evidenced by human body burdens indicate that any of those persons is going to develop a disease as a result of that exposure.
Q. Okay. Now, I have the letter you wrote back in April of 2000 to the ATSDR. Do you remember that?
A. I do.
Q. Did you have any help in writing this letter? Did you write this letter?
A. Yes, I wrote that letter.
Q. Okay.
A. A few people moved some comas.
Q. Sir?
A. A few people moved some comas, but it is almost exclusively my work.
Q. Okay. And what did you review -- this may be such a broad question you can't answer it. What all did you review in order to write this letter and it was the comments to the --
A. Believe me, I know what it was the

comments to.

Q. Health consultation.

A. Dated February 14th, 2000.

Q. Right.

A. Which, for the record, has never been finalized, nor have my comments ever been addressed.

Q. That's what I was going to ask you.

A. They keep promising they are going to within the month.

Q. So they haven't responded or corrected anything?

A. No. The procedure would be that they will take into consideration my comments or Solutia's comments, other comments which they have received, which I am sure have been many, and they will, in their view, appropriately respond. They will revise the document as they believe appropriate in response to those comments. They will then publish a final version of the document and will publish, as an attachment to that, the responses

to all of the comments that they received.

Q. All right. With that said, let me ask you a couple of things and I'll show it to you, but you may know this. You may have this memorized.

A. Well, and it's a lot of the things we have already been talking about.

Q. I think that's right. You mention here, and, again, when you refer to page five, the one I have doesn't have the same page so it may be off. I don't know why.

A. You may have gotten yours off the internet or something.

Q. I may have.

A. There's an internet version and a hard copy version.

Q. When you mention page five, second paragraph, it says "Solutia does not believe it is appropriate for ATSDR to speculate about the reduction in the ninety-fifth percentile of PCBs in blood

a person is exposed in a manner quote 'typical' unquote for the U.S. population. In the first place, we do not believe it is appropriate to rely on the draft Tox Profile for PCBs."

And my question, basically, I read that for this reason: at that time that you wrote this letter, the Tox Profile was a draft?

A. That's correct.

Q. It has now been peer reviewed and finalized; correct?

A. Well, it has been finalized. We can quibble about what peer review means, but they say it has and I say it hasn't.

Q. Okay. The only reason I say it has been peer reviewed is they have.

A. It has been reviewed and it's final, no argument about that.

Q. And I guess my question to you is when you -- did they change anything about the Tox Profile that you were quote "criticizing"?

A. Yeah, I think nowhere in here do they say that the normal background range is ten parts per billion in blood. They talk about this average.

Q. Okay.

A. But as far as I know, they haven't -- in the draft, they specifically said the normal human range is now ten parts per billion already. There are no data to support that. They don't say that in here. They don't say it in subsequent health consultations that they've issued with regard to Anniston.

Q. So the criticism that you talked about is not in the final version?

A. Well, but this -- well, I don't know because I haven't -- I think that's correct and I don't remember exactly what the -- I know where you are. I was going to say my letter is not addressed to this document, but you know that.

As far as I know, and I don't remember the exact quote from the draft

profile, but whatever I was objecting to I think has been changed in the draft profile. I don't recollect anything in here which would trigger that comment, let's put it that way.

Q. Do you think today you would not have that same comment?

A. I would have the same comment if they tried to say the background range is ten parts per billion without justification, but I may not refer to the Tox Profile and them quoting that for that premise for their thing. I mean, if they have data, fine, but my objection was they don't have any data. That was my real objection and that was evidenced because they didn't cite the literature. They cited the draft Tox Profile.

Q. Do they have data that mean serum levels range from point nine to one point five?

A. In recent studies, I think they probably have a couple of studies that

they averaged to get those numbers out.
Q. Now, you also have some criticism here about the extrapolations. "A suggestion that back extrapolations can be used to estimate historical PCB body burdens in Anniston is over simplified."
A. I don't remember saying that, but okay. I'm glad I did.
Q. "This discussion must be expanded to elaborate on the uncertainties involved in and introduced by this process, speculative process," you call it, and then you talk about "in the first place, PCB half lives are congener specific." What are the half lives of PCBs?
A. Well, let's go to what is a half life.
Q. Okay.
A. All right, whether you're talking about a chemical or a pharmaceutical, whatever, if you put something in your body and have some measure, then, of the level of that thing in your body,

whatever it is.

Q. The dosage amount?

A. The dosage. Well, no. A bio-marker of the dosage. You may not know exactly what the dosage is, but some level -- well, it could be a blood level, it could be a breath level, it could be a level in your urine. You have some measure of how much of that material got to whatever --

Q. Ten parts per billion?

A. Yeah, whatever your measure, okay, your body basically takes everything that goes into it and does something to it and makes it go away. Some things go away faster. Some things go away sooner. Most of the things that go away go away in a concentration-dependent manner so that you can measure, for instance, if I take an aspirin and I can measure aspirin in my bloodstream, right after I take the aspirin, at some point, the concentration of aspirin in my bloodstream is going to be half of what it was, all right, and

that's a half life.
Q. Whether it be hours or days or whatever?
A. Days or minutes, whatever. And then if you start at that point, then one half life later, you're only going to have a quarter of it left so it's basically a way of measuring how quickly things are removed from your body.
Q. Okay. With that said, can you say what the half life is of PCBs in general?
A. I do not believe you can.
Q. Okay and that's because --
A. That's because there's no such thing as a PCB. There are two hundred and nine different things called PCBs that each one is going to behave differently in a human body and an animal body and they're each one going to have an unique half life.
Now, you can -- and people have -- I'm not so naive as to say that

nobody's ever tried, in a given population, you can look at blood levels and make some generalizations about half lives but those generalizations are population specific.

Q. Okay. And what is --

A. So if you're looking at capacitor workers and you've got a hundred or a thousand capacitor workers you can get some kind of average half life for, you know, lower chlorinated PCBs and higher chlorinated PCBs which is what has been done, as you well know, but those are highly exposed people and the half lives may differ depending on whether they are highly exposed or lowly exposed.

There's all sorts of averages in there. You can't say on the individual person that you can do that calculation. You can only do it on a populations basis, if at all and on a population basis, I believe with a similar exposure scenario.

Q. So do you have any -- what is the half lives of the general population?
A. I don't know.
Q. You have no way of knowing?
A. I know what has been measured in capacitor workers.
Q. Which is?
A. I think for the lower, I'm guessing it was one to two years and for the higher, it was seven to eight years, but that only applies to those capacitor workers and it applies on a Hugh's average and, you know, you can't do the calculation. You can have the concept. I don't have a problem with the concept --
Q. Right.
A. -- as long as you can check for background first.
Q. Right.
A. If you know someone who is exposed and you know that that exposure blood level is decreasing over time, then you

can make a general argument that, you know, correcting for background, that some person's level may have been higher. But I don't think, with PCBs, you can do that with any certainty whatsoever.

Q. Can you take -- do you know the half lives of various congeners?

A. No, I don't think anybody does.

Q. Has that ever been studied or measured?

A. I think those studies are beginning. I think Dr. Hanson talks about the fact that those studies are beginning, but I think, you know, I mean, they haven't been done. And until you have two hundred nine of those studies repeated with some certainty around those numbers, you're not going to be able to do it. I don't think anybody will ever do it because it's too complex and it doesn't really matter.

Q. Okay. When you say doesn't really

matter, it would matter if it were something you could use to extrapolate, I guess?

A. It would matter if someone were going to get sick depending on what the outcome would be.

Q. Okay. Again, we go back to the risk associated with exposure period?

A. Right. I mean, why are you doing a calculation? Presumably, if you're going to do it, it's to try to say that, at some point in time, that person had a higher level which means something.

Q. That's exactly what I --

A. Well, yeah.

Q. But you don't agree that that's possible or feasible or correct?

A. Because of what my view of the what the literature says about the health effects of PCBs, no, I don't know that, no matter what number you get to, it matters, but I think any number you get to today is going to be so uncertain that

it's meaningless anyway.

Q. One other point in this letter that you wrote, and you're talking about the note that they say that six hundred forty-eight children live within a one-mile radius and, of course, you disagree with the number. One time, it says six forty-eight and one time, it says six fifty-eight or something, but anyway, "More important, the significance of selecting a one-mile radius around the facility is unclear. Such a circle includes large areas which are in the drainage patterns from the facility in which PCBs have not been detected in soil above one part per million level defined by the EPA as clean in other areas."

When you said that, that raised the question I asked you early on which was in the cleanup of the areas that we're talking about in the proposed agreement. Are you going to exclude the areas or are you going to try exclude the

areas that are not in the drainage, as you call it, the drainage pattern?

A. Well, I don't think we're going to -- we're not going to exclude them. Can we go back to what we've already done? Let's talk about the consent order and what we're doing.

Q. And I think I have that.

A. The areas that are not in the pathway were not excluded by definition, but our sampling program focused on the areas in the drainage pathway and, again, what we talked about earlier, and as we and the EPA got more confident that we were moving away from areas associated with the drainage pathway and we weren't seeing PCBs, that our sampling could be curtailed inside those areas.

So I think, going back to what I think the original question is, I believe that we, with the oversight of the EPA and whoever else is appropriate will continue to try to focus our

sampling and our remediation efforts on the areas associated with the drainage pathway because that's where we believe and are very confident that the PCBs associated with our form of manufacturing are, in fact, located.

Q. Because I assume you don't agree that if there is a high level of PCBs in a soil outside the drainage area, you don't agree that it got there by virtue of the normal exits from the plant?

A. That's correct, I do not, no.

Q. That it got there some other way?

A. Correct.

Q. Fill dirt?

A. Primarily. That's our working hypothesis, yes.

Q. What about areas that are outside the drainage basin that have trees, for instance, in it that have PCB levels; how do you explain those?

A. I don't know that I have seen the data to explain them, but I don't want to

speculate. You know, I know Dr. Hermanson has been out there measuring tree bark. I don't know what that means, frankly.

Q. Okay.

A. Because we don't know how old those trees are. We don't know what contributes to getting PCBs on tree bark. I don't know what calculating, on a lipid basis on tree bark does. There's a lot of questions about that, but I don't have a ready explanation.

I mean, obviously if they're there and they're truly there, there is some explanation of how they got there. I don't have a ready explanation for that and I don't know why these are that are located outside the drainage pathway and I don't think, if they are in tree bark, I don't think they got them from the drainage pathways anyway other than possibly some blowing dust or something. What I'm saying is I don't think they're

coming up through the roots and getting up to the bark.

Q. And I guess the question -- that goes back to my question earlier, if a piece of property is outside the drainage basin or flood plain or whatever you want to call it, can the levels of PCBs in that soil have gotten there through the air or particles being traveling through the air into that property?

A. No, I am convinced that is not true. At levels that are in the range we have been talking about, the one part per million or ten parts per millions, I mean if you are going to get down to the parts per trillion, then there may be some air transport, there may be some dust transport, but at levels that are going to be significant for cleanup operations, I don't think the air pathway is significant either through air itself or through dust.

Q. What way would the property outside

the flood plain have gotten a large -- how would that have gotten the fill dirt, as you call it, or where would the fill dirt have come from or do y'all have any knowledge about that?

A. Well, there's some apocryphal knowledge. There's some speculative information. You know, I don't want to be the carrier of tales, but, I mean, it's certainly my understanding that the foundries, sand piles, used sand piles were pretty much freely able to anyone in Anniston, anyone to come with a drum or a bucket or a pickup truck and take them away to use it as fill.

And we also, I mean we've got apocryphal stories of people going down into Snow Creek and taking sediments out of Snow Creek and mud and stuff to fill a place in the yard or something. Usually, it's been people closer to the creek where that was kind of -- seemed like a natural thing to do. But that's, I

guess, from, you know, hearsay and speculation, but that would by my answer to your question.

Q. Now, I think I have the map and it's not a very legible copy. Is that the map that we're working on with respect to the areas that --

A. No. The map you're looking at is basically a map of the -- now, these are actually the areas that we own, these are our properties.

Q. I'm looking for the map that describes the areas and I think you said one, two, three, four, five, six, and I --

A. I can use a break --

Q. I'm sorry, yes, let's have a break.

A. I'm just saying I could use a break anyway. Let me get a drink of water and let me leaf through this and see if I can find it for you. I mean, I can't guarantee it's in here, but it certainly should be.

Oh, this is the consent decree (referring to document). It's not going to be in the consent decree. It's going to be on the Administrative Order of Consent from October of 2001 -- the EPA Administrative Order of Consent. It's not going to be in here. It's going to be on there (referring to documents.)

(Whereupon, a short recess was taken at this time.)

Q. We'll go on to a couple of other points with your letter, Dr. Kaley. The blood dioxin measurements that you refer to?

A. Yes.

Q. You don't expect, as I understand, to find -- well, tell me, do you find dioxins in PCBs?

A. No.

Q. Okay. Do you find dioxin-like congeners in PCBs?

A. Well, you're using a term I loath.

Q. What term would you rather use?

A. I don't know, age-responsive. I'll use dioxin-like because -- under protest -- but are you talking about PCBs that have dioxin-like properties or are you talking about other compounds that are dioxin-like in PCBs?

Q. I'm talking about what I understand to be called coplanars, which is what?

A. Okay, and it's too bad we don't have our model, because we would know that.

Q. What are coplanars?

A. PCBs are made up of two benzene rings and those rings, as we talked about during break, PCBs are molecules not just sitting there rigid in space. It's twisting and bumping and spinning and things are happening.

A benzene ring is a flat molecule. It's made up of six carbon atoms and kind of like a hexagon and it's very flat. If you were able to look at it spacially, it would be flat. If you hook two of those together with a

carbon-carbon bond, they'll spin in reference to each other constantly. And if it is possible and they do get into a configuration where they are flat in the same plan, then they are called coplanar PCB molecules.

Q. Do they still rotate?

A. Yes, it's just whether they can get in there. What's important about that is that there is, in animals and humans, a physiological -- what's called a receptor called the AH, capital A, capital H, into which a molecule called the TCDD or dioxin can bind and trigger, arguably, a set of physiological events.

The receptor is structured such that only things that are coplanar, that are flat, will get into that and bind into that receptor. A dioxin itself isn't one of these flat molecules. So if you have a PCB which can get into this flat configuration, and it does so at a time that it is in proximity to that

reception, it can bind to that receptor. That's why it matters whether they are coplanar or not.

Q. That's why some people call it dioxin-like?

A. Yes, because the theory is, and it is a theory, is that once a PCB congener can bind into that receptor, it could then trigger those same responses that dioxin would, although at much lower levels or much lower potency.

Q. Okay. And why is it that you do not agree that that can occur; that is, that it can bind to the AH receptor?

A. I totally agree that the binding can occur. I do not disagree that the binding can occur. What has never been shown in the laboratory is all the rest of that cascade of physiological or toxicological events that have been shown for dioxin. No one has ever really shown that few, if any, of those have ever occurred by the binding of a PCB molecule

into that receptor. Clearly, the binding occurs. There's no question about that.

Q. There are certain PCBs that can bind to that receptor?

A. Yes.

Q. Which ones are those?

A. Well, there are four PCBs that can totally get into this coplanar structure or format and have chlorines in the right places. Do you want their numbers; is that what you want? Do you want their chlorine substitution patterns? I mean they are numbered eighty-one, seventy-seven, one twenty-six, and one sixty-nine.

Q. Okay.

A. Those are four totally coplanar PCBs.

Q. All right.

A. They were present at extremely low levels in our products and they are present at extremely low levels everywhere else.

All right, there is a another group of about seven or eight that have one chlorine next to this bond that we're talking about and they can't get totally flat but they can get kind of flat and they can get flat enough that they'll bind even weaker than the PCBs so they can get in there and they'll bind a little bit, but any effects they have would be totally minimal and I mean one hundred five and one eighty, and one thirty eight. There's some others. I don't have them all memorized, but there's about a dozen altogether that are coplanar.

Q. So the part you don't agree with is that once these coplanars attach to the AH receptor, the effect is not the same as you would have had if you had a dioxin attached to that AH receptor?

A. I think that has never been shown.

Q. Is that, in simple terms, enough, at least, for me to understand?

A. I don't want to make it sound like I say it's impossible, but I'm saying because they bind so much more weakly and because there are other things going on with PCBs, the most important of which is that you don't ever have this single congener or a group of these single congeners out in nature or in a body. You've got whole bunches of other PCBs out there which are trying to bind and which are binding to other things and which are interfering with the binding, etcetera, etcetera.

So until you go into the laboratory and, number one, show that that congener can cause whatever effect-- cancers are a biggie -- you know, dioxin in animals causes cancer.

Q. That's a known?

A. That's a known, all right, although there's an interesting dioxin study going on at EPA that EPA is going to have big problems here coming up because they're

doing their own study and it isn't causing cancer nor is the PCB they're testing, so we'll see where that comes out.

Q. Is that going to come out before October?

A. No, they're sitting on it, believe me. I'm hoping it will.

Q. Okay, just asking.

A. And on the other side, we've got PCB studies that if you feed Aroclor 1260 to rats in the laboratory, you get cancer. What we don't have is taking that one single congener out of PCBs, feeding it to rats, and ending up with cancer, and until you have that, it's all theoretical. And that's exactly what EPA is trying to do in their experiment and it's not working out for whatever reason.

But even if you do that, you're still dealing with that single congener. You're not taking into consideration all

the other things that can go on, and I don't know whether you've had the agonist-antagonist lecture and all that stuff, but some PCBs apparently lesson the effect of dioxin, so it's just very complicated, and until we really understand and can demonstrate that PCBs can do all these things that are alleged to dioxin, then we're on very shaky ground.

I mean, you know, you say who cares? Well, I think the problem is and who cares is that people use this theoretical construct to try to draw PCBs into the whole dioxin health effects discussion, whether it be animal or human or whatever and there's no scientific basis to that.

Q. So we all know that dioxins, in and of themselves, do cause cancers in humans?

A. No, no, no. Rats.

Q. There are animal studies that --

A. That show that TCDD, a single -- not like PCBs, there are, you know, seventy-five dioxins.
Q. There's one dioxin and it causes cancer in rats?
A. Yes.
Q. What's the difference between that study and the study that shows PCBs cause tumors or cancers in rats?
A. Well, I'm not sure what your question means. I mean, it's a different chemical. I suppose it's as DDT causes cancer in rats or something else. The problem really is that dioxin does it at so much lower levels.
Q. Okay. That's what I'm saying. We know that there are animal studies that show an association with --
A. I think in animal studies we've got all that controlled. I'll go with show.
Q. PCBs show that there's cancers in rats?
A. Yes.

Q. We also have studies that show dioxins cause --
A. A dioxin causes cancer.
Q. What my question is, I guess, is, all right, how can you take the giant leap from the dioxins in rats causing cancer in humans and you can't take a giant leap with respect to PCBs causing cancer in rats to humans? Why can't you take the same leap?
A. I don't take the first leap. I would never take the first leap. You can't take either leap.
Q. So you won't agree that, based on the animal studies of dioxin causing cancer in rats that that proves or shows that dioxin causes cancer in humans?
A. Oh heavens, no. I mean, we would have exactly the same discussion around the dioxin-human lecture that we do with the PCB. What I was trying to say is that TCDD causes cancer in rats.
Q. Okay.

A. PCBs -- A PCB mixture, Aroclor 1260 causes cancer in rats, but the in-between is one teeny component of that Aroclor 1260 has never been tested to determine whether it causes cancer in rats, so we can't make the leap that, you know, because that little, you know, we don't know the mechanism of really how dioxin causes cancer in rats or how PCBs cause cancer in rats.

What the theory is is that if you've got that little teeny bit of, let's say PCB 169 in Aroclor 1260, if it binds to the receptor, it's going to cause cancer by the same mechanism that dioxin does, so everything you can say about dioxin, you can say about that compound. We don't know whether it causes cancer in rats so we can't make that -- that's the leap we can't make. Humans are out of picture at this point.

Q. Okay. That's what I'm trying to get at. If you don't agree that there's

quote "proof" that dioxin causes cancer in humans --

A. Okay, and I don't.

Q. You don't. Then what difference does it make if we prove that the dioxin-like congeners attach to the AH receptor and cause the same risk to humans as dioxin does?

A. There are numerous regulatory implications of that assumption. It can drive cleanup levels to unreasonably low and unreasonable expensive levels. It can cause the EPA to regulate chemicals in our food supply at, you know, ridiculous costs to the economy. There are numerous --

Q. Ramifications?

A. Ramifications, that's the word. There are numerous ramifications of that assumption that, until it's proved, we should not be making those leaps.

Q. There are quote "scientists" out there that do come to the conclusion, I

assume, that dioxins do, in fact, cause cancer in humans.

A. Yes.

Q. Are there clinical studies out there similar to the studies that I mentioned earlier, the Brown and Bertazzi, that have found cancers associated with dioxins?

A. There's a body of dioxin literature which some people interpret as being demonstrative that dioxin causes cancer in humans.

Q. Is there the same problem with those studies and that literature, is there the same problem with that literature that there there is with the PCB; that is, that one study may show a certain site of cancer in one site and another study won't show that; that is, inconsistency? Is there the same problem with those studies?

A. That is a minor part of the problem with dioxin studies. Interestingly,

dioxin studies do have some consistency in that they seem to show that there is a very weak elevation in what they call all cancers combined, that it really doesn't cause a cancer in any particular site which is problem number one because there's no other compound that's known to do that.

Q. Okay.

A. So this whole all cancers combined, it just doesn't make any sense. You know, one of the other criteria that I'm sure you've heard people talk about is biological plausibility. It's not biologically plausible that dioxin could cause these teeny elevations, although statistically significant, and I'm not going to play that game. They are statistically significant in all cancers combined without looking at any particular cancer.

And there are huge problems with confounding in those studies because

most of those were studies of workers in chemical plants where there were lots of other carcinogens. So there is a body of literature, some of which can arguably stand for the proposition that dioxin causes cancer in humans. It's clearly been interpreted that way by the regulatory agencies, at least EPA, and I think there are problems with it, so I'm not going to buy into it, but the literature is clearly there.

Q. And I assume that the EPA's assessment, you don't agree with; that is, that it causes cancer in humans? Dioxin?

A. Right, I do not agree with the EPA's attempts to label dioxin a known human carcinogen.

Q. Okay. So the letter, when you talk about the TEQs, meaning what? What are the TEQs?

A. Well, one of the outgrowths of the fact that some people believe that some

PCBs have dioxin-like activity is that you can, by looking at, you know, binding to the receptor or some other end point, come up with a number, a ratio of the potency of a particular, for instance, PCB to TCDD so if TCDD -- you can measure how strongly something binds to this receptor. If TCDD binds, you know, strength "X" and this PCB binds at a strength one tenth of that, then they have this theoretical construct called toxicity equivalency factors TEQs to relate those potencies or those strengths of toxicity.

If you have a mixture of compounds and you take the concentration of each compound and multiply it by its concentration in that mixture, you get what are called toxicity equivalents with a TS and so those are TEQs which is "T" from toxicity and "EQ" from equivalence, TS, not CE, and that theoretically relates the toxicity of that whole

mixture back to dioxin.

Q. And what they're measuring, as I understand, is the eleven or ten or whatever the number there are that are quote "dioxin-like"?

A. Correct, the ones we talked about earlier.

Q. Which included the four or five you said that were truly dioxin --

A. Right, truly coplanar, right.

Q. Coplanars. And those that are the little swiquets.

A. The little swiquets.

Q. Okay. And they come up with this TEQ?

A. That's correct.

Q. Do you place any significance in the level or the TEQs in humans? I mean, do you put any significance into that?

A. The TEQ, the whole toxicity equivalent factor, TEQ construct was developed as an interim procedure and it's still an interim procedure by EPA to

help them economically assess risks for cleanup sites.

If you have a complex mixture of dioxins or other, you know, dioxin-like materials, arguably, to do a risk assessment on that site, you would have to go in and measure every compound, have toxicity studies on every compound, and assess independently the risks of every compound in that mixture, and then somehow add all those up. So everybody -- and I would agree that's -- we don't have the toxicological information. It would be years away and very costly to get the toxicological information.

The analytical costs would be extreme, etcetera, etcetera. So EPA and others come up with this scheme where okay, let's look at this mixture, let's look at these toxicity equivalency factors which you get from animal studies or test tube studies or whatever and give us this TEQ for this site or this soil or

this residue which gives us some feel of how that site should be cleaned up and we'll relate that to dioxin and we'll clean up to that dioxin level rather than having to do it for each individual compound.

I think it has some justification in that scheme. Now, I draw two lines. One is I think that scheme is entirely inappropriate and premature for PCBs.

Q. Why?

A. Because we don't have any of the information that we've been talking about, the toxicological information that says PCBs even behave in those manners.

Q. That's the same thing, you don't think there's information to show that the coplanars or the dioxin-like PCBs cause any health effects?

A. Right, even in animals.

Q. Even in animals?

A. Even in animals to do that.

Secondly, along the same -- well there's actually three. The next disconnect is we have been doing PCB risk assessments and PCB cleanups for decades based on looking at PCB mixtures as we know them and we find them and we love them.

Q. Total?

A. Total PCBs. Things are getting cleaned up. Things are moving along. The sites, you know, there's no evidence that any site that's ever been cleaned up that way remains in any kind of theoretical or real risk. It's worked fine and it's just so much more efficient.

The other disconnect is that to take this theoretical construct and try to apply it to human body burdens, I believe is totally inappropriate and others agree with me. And the EPA doesn't agree with me. The EPA is trying to do it. And there's no justification

to say that a body burden on a TEQ basis has any relationship whatsoever to reality.

It's a convenience to help us gets sites cleaned up. It was never designed to, nor is it robust enough to talk about threats to human health based on these TEQs.

Q. And you say others agree with you. Do you have any particular reference to others who agree with you?

A. No, I mean, it's an ongoing discussion.

Q. Obviously, there are others that disagree with you?

A. It's an ongoing discussion. It's one of the real discussions that's, frankly, holding up this draft dioxin reassessment that's making its rounds to government agencies because it's a huge question.

Q. Well, in your statement to ATSDR, you talk about specifics and I think you

mention someone, their ages and you talk about the fact that because of their age, they would have a significant TEQ. Maybe I'm reading that wrong.

A. I don't think I said that.

Q. Maybe I better read it correctly.

"Examination of the birth dates clearly shows that this is an elderly group of persons. The youngest was fifty-one at the time of sampling and analysis while the oldest who had the highest PCB level, total PCB level was eighty-four. The significance of the ages of these people will be discussed. Person one has already been discussed. This person is a former Monsanto worker. He also has the highest level of TEQs among the group. Examination of the data sheet for this shows that his TEQ level is dominated by TEQs for Penta and hexa dechlorinated dibenzofurans which is exactly what would be expected for someone with an elevated PCB level."

And I misspoke while ago. Why would he be expected to have a high TEQ associated with dibenzofurans?

A. Okay, this person was a worker, a long-time worker at our plant. He had obvious exposure to PCBs. He had an elevated PCB level. The PCBs, as manufactured by Monsanto, had trace levels two to five parts per million of chlorinated dibenzofurans as impurities as measured, so somebody exposed to high levels of PCBs is going to, you know, logically, at least, maybe not for sure, but possibly have also elevated levels of chlorinated dibenzofurans, but not dioxins, and that's exactly what this guy shows.

Q. And you mention, you go forward and you say low levels, typically a few parts per million of PCDFs were produced; that's what you said?

A. Right.

Q. But you're saying that there were no

such -- there was not a same level of PCDD; is that right?

A. That's correct. In fact, they're not measurable or not measured. They have never been reported of dioxins and PCBs as an impurity of manufacture.

Q. So if there are -- so how do you explain the dioxins found or the dioxin-like congeners found in the blood levels that we have measured?

A. Okay, now --

Q. Are we talking two different things here?

A. You start talking dioxins and then you started talking dioxin-likes.

Q. Okay.

A. I mean, there's three or four things we need to keep straight. One is there are a group of compounds called dioxins, all right, and the most well known of which is this TCDD we've been talking about. There's a related group of compounds called polychlorinated

dibenzofurans or on short, furans.
Q. Right.
A. All right, there's another group of chemicals called PCBs.
Q. Okay.
A. So we've got these three big groups that we're all going to be talking about. Now, within the PCB group, there's this little teeny twelve-member little subgroup called dioxin-like as you refer to it and others are the coplanars. So your question was dioxins or coplanars and they're separate answers to those two questions.
Q. All right, that's maybe my confusion in reading this letter when you say PCDDs or dioxins are not produced as by-products for the manufacture of PCBs, nor are they formed by thermal stress of PCBs.
A. Right. That's that one group of specific compounds, right.
Q. "Therefore, the PCDD or dioxin levels

in persons in this community cannot be associated with their potential exposure to PCBs."

A. Right.

Q. "In the second place, the magnitude of dioxin levels in this person clearly suggests that she has had some unique exposure to dioxin assuming, of course," and so on. You're referring, as I understand you now, you're referring here to the fact that they're talking about dioxins as opposed to coplanars or dioxin-like PCBs?

A. This was a separate set of analyses for dioxins and furans and PCBs.

Q. Okay.

A. But I was only addressing, in these comments, the dioxin and furan issues and they are separate issues when being discussed around PCB questions. PCBs were out of that whole discussion, other than the fact of as a potential source of furans in that one person. But other

than that, all I was talking about was the dioxins themselves and the furans themselves.

Q. So you weren't talking about, in this context, you weren't talking about the coplanars; is that correct?

A. That's correct, I was not.

Q. Did ATSDR even mention the quote "coplanars" as it relates to the TEQ?

A. As I sit here today, I don't recall. I think those data were available to them. I don't know whether they tried -- I don't recall.

Q. Well, the reason I'm asking that is, in the same paragraph, you're mentioning the TEQs and the dioxin and furans, and that's what's confusing me some because I --

A. Because the TEQs were originally developed only for dioxins and furans. TCDD is the prime example. It's the ones in which everything else is -- it's a reference compound. That's the word I'm

looking for. It's the reference compound and it's defined as having a toxicity of one, just unitless, just one.

Q. All right.

A. There are, I'm going to say, seven other dioxins and I think ten furans that also, like TCDD, bind to the AH receptor and, in some cases, have been shown to, in other cases, are assumed to have dioxin-like activity so they have TEFs associated with them also, all right. And that's what -- when I was talking about cleanup levels and the development of the TEF concept, it was only four dioxins and furans, all right.

The attempt by the EPA to roll those coplanar PCBs into the overall structure is fairly recent, okay. That's not been done until this latest dioxin reassessment, so when I'm talking here about the TEQs, I'm talking about the TEQs for the dioxins and furans in those, not the TEQs for the PCBs. That would be

an addition onto the TEQ if we were talking about those.

Q. And I think you're referring to table six in that paragraph; right?

A. Yes.

Q. And in your responding to or criticizing or however you want to determine it in this February 14th, 2000 health consultation?

A. Yes.

Q. And in table six what they say -- let me find it. I had it. Table six says total blood PCBs, dioxin toxicity, TEQs and levels and year of the birth, blood dioxin/current/coplanar PCB analysis.

A. Right.

Q. That's what I'm confused by.

A. If you look at the table, the first column is reference number for the person.

Q. Right.

A. The second number is total PCBs.

Q. Right.
A. Third and fourth column are the sum TEQs for dioxins, furans, and PCBs in blood where the third column is without the PCBs included and the fourth column is with the PCBs included.
And when I'm talking specifically here about that, you know, maybe I wasn't clear here, but I was talking primarily about the withouts. I'm talking about the TEQs from the dioxins and furans, not from the PCBs.
Q. All right.
A. Now, that person, because he has higher PCBs, I think it's number one, obviously, if it's the highest, is going to have, with the PCBs, a higher number than the others also because he's got more PCBs, so that would just make sense. But that comment was really addressed to this without the PCBs column --
Q. Okay. And then you mention the --
A. -- because I think I say one

seventy-nine, don't I, in there? I thought I just saw where I said the person --

Q. I think you did somewhere.

A. Anyway, that's the confusion because you can do TEQs with PCBs or TEQs without the coplanar PCBs and my specific comment there was addressed to just the dioxin and furan analyses part of the --

Q. Person nine, the magnitude of OCDD level, in person nine, clearly suggests that she had some unique exposure to dioxin?

A. OCDD, octidioxin, which is -- it counts, but it's the weakest of the lot.

Q. What was her level of PCBs?

A. Of PCBs?

Q. Right, number nine?

A. Her total PCBs was one hundred three parts percent billion.

Q. And her TEQs?

A. Was sixty without the PCBs and two hundred ninety-two, but the octidioxin

doesn't account for much TEQ because its factor is one ten thousandth so you multiply its concentration by one ten thousandth to calculate a TEQ, so it doesn't really contribute much.

Q. Why do you say that she had some unique exposure to OCDD?

A. Because her OCDD level is very high.

Q. Okay.

A. Compared to everybody else in the population and compared to national backgrounds and compared her other dioxin and furan levels. It was just out of whack with the pattern you would expect to see.

Q. How would you explain that?

A. How would I explain it? I would explain it as a lab analytical error. It could be -- OCDD is a product of combustion. I mean, if for some reason, she was around a lot of forest fires or burned a lot of trash or something, you could do it, but I think it's probably an

analytical error. It's just sticks out so badly. I can't -- you know, in my comments, I couldn't speculate on it. I'm just saying it sticks out.

Q. All right. On page fourteen of your letter, I think you're talking here about some, I guess, soil samples, yes.

"Solutia suggests that the agency avail itself of all avenues to ascertain the conditions under which samples were collected by agents of the plaintiffs' attorneys. It is Solutia's understanding that many of the samples were collected in areas calculated to maximize potential soil levels such as downspout." Can you tell me who you're referring to?

A. To what plaintiff's attorney I'm referring to?

Q. No, agent.

A. I mean, those data were all supplied by the plaintiffs' attorney in the Abernathy litigation, so I'm talking about whoever collected the samples.

Q. You don't know particularly who that was, not attorney but --
A. I'm tempted to say Mr. Bonner or Dr. Bonner, but I don't know that for a fact. I could be wrong on that. I'm tempted to say that's who it was and, again, let me -- I'm not saying he did anything wrong.
Q. I understand.
A. I'm just saying it wasn't an average value. My understanding was that they went to areas on the property where they expected the levels to be the highest.
Q. Well, you go on to say that "our understanding is based on the deposition testimony of one of the persons who performed soil sampling for the larger of the two plaintiffs' groups. For instance, we will provide relevant sections of that deposition to ATSDR upon request."
A. Yes, and I don't think that was ever requested and I don't think we did.

Q. You don't know who you're referring to?
A. I think it was Dr. Bonner.
Q. Dr. Bonner?
A. I think so, but I could be wrong.
Q. Okay. Now, any area you refer in here as under other pathways, you say that in Monsanto, in 1970 or '71, Monsanto became aware that hogs were being raised on or near Monsanto property on an area that had been used for waste disposal. Can you specify what area you -- do you know what area you're talking about?
A. It was somewhere on the south landfill. More specifically than that, I don't know. That's my understanding.
Q. The hogs rooting around in this secluded area were possibly being exposed to waste materials. Do you have any information as to whether or not these hogs were analyzed as far as PCB levels?
A. I believe they were not.

Q. Was there ever any time that logs were analyzed?
A. My understanding is I think it's covered by people's testimony. My understanding is that a hog was found dead on Solutia property, that its PCB levels were measured and found to be elevated, and that, based on that, people from the plant found that there were other hogs on the property and that those hogs were purchased but were not tested.
Q. Was there any analysis done on the hog that was, in fact, the initial hog on the liver?
A. I don't know what was -- I do not know what was specifically tested. I don't know. I think the fat was, but I don't know whether the liver was or not.
Q. And I think y'all found the map or a map?
A. Yes.
MR. KELLY: Can we go off the record?

MR. RODEN: Yes, okay.
(Whereupon, a short discussion was held off the record at this time.)
MR. RODEN: Make a note we're going to mark Exhibit No. 1 and we're going to substitute it for a copy.
(Whereupon, Plaintiff's Exhibit No. 1 was marked for identification.)

Q. All right, Dr. Kaley, just for purposes of identifying, just tell me what we are looking at? What is this?
A. This is a map supplied by the Environment Protection Agency which designates by number with one exception, one of them is by letter, areas in the north and east of the Monsanto plant which were to be tested for -- which residential properties were to be tested to determine if PCBs were present in those properties in five point composite

samples above ten parts per million.
Q. And is this the same legend that has been attached to the proposed order as pending?
A. This is an attached -- this is part of an attachment to the proposed consent decree, but it is -- what's really attached to the proposed consent decree is the administrative order of consent dated -- I think it was October 3rd, 2001 to which this is an attachment or an exhibit, so it's an attachment to an attachment.
Q. Okay. What was the purpose, again, of this particular map?
A. The purpose of this map was to delineate and prioritize areas for sampling for Solutia under the terms of the consent order.
Q. And then the proposal, its function is the same under the proposed consent decree?
A. I don't think so. I think -- well,

I don't know that the Anniston PCB site enclosed as defined in the proposed consent decree is exactly the same as that. I do not believe it is. I think it is a much larger area, but the consent order is going to be rolled into the consent decree and will continue to be executed so, in reality, as far as I know, all this still addresses properties which will be sampled the ten part per million removal action. I don't know that this drives either the sampling or the additional sampling for the consent decree.

Now, I do know that the properties identified in the sampling under the consent order which have greater than one part per million, assuming everything is approved in the courts, etcetera, will be cleaned up under the terms of the consent decree when it's executed. Sorry, that got very confusing.

We have sampled -- the consent order told us to go out and sample residential properties in these areas.

Q. And you sampled how many?

A. I'm going to say nine hundred. I could be wrong. Mr. Branchfield would know. I'm going to say nine hundred, okay. We did five-part composites, okay, with the primary goal of identifying those with greater than ten of which there were, I think, twenty-eight or something like that, mid-twenties, and those ten were to be cleaned up in accordance with the consent decree. We've done that on thirteen, twelve, or so. I think it's twenty-five. We've done thirteen. Twelve, we were access by the plaintiffs' attorneys or the owners' attorneys.

Q. Okay.

A. In doing that, some of these properties were found to have one to ten parts per million PCBs. The consent

decree will address cleaning up those properties and those properties with levels between one and ten will be cleaned up under the terms of the consent decree.

What I don't know is, for sure sitting here, is if additional sampling is required outside of these areas. I don't know that as I sit here.

Q. So you don't know if the consent decree expounds on this particular area?

A. As I sit here, I do not know the answer to that.

Q. But my question is -- assume just for the sake of this question that the map, whatever the map is, whether it's this map or another map, it is what it is and you're saying the sampling will be conducted only in these areas; is that correct?

A. Certainly, under the consent order, that is correct.

Q. And it would be true on the consent

decree, it would follow a map whether it's this map or another map?
A. I believe that would be correct.
Q. So if a piece of property is outside these zones, would they ever be tested under the consent decree?
A. You're stretching my knowledge of the consent decree. My belief is that they would not be.
Q. Okay.
A. Now, can I make just one more addition just to clear things up?
Q. Sure.
A. There is one additional area that is not shown on this map. It is called Area OLN, all caps, which stands for Oxford Lake Neighborhood and that's a little area down by the Oxford Lake softball complex down off Highway 78 so that's also included in the consent order, but it's not on this map.
Q. What prompted that?
A. The discovery of a couple of yards

there that had PCBs in them.
Q. Was that through some litigation?
A. No, it's part of our investigation along our -- well, I think it was actually prompted by a request by one of the residents there to have his property sampled because we had been doing work along that neighborhood and my recollection is he had taken sediments from the creek and put them up in his yards, so we tested an additional group of properties down in that area.
Q. What is the significance of the zone "F"?
A. I don't recall right offhand. I think that's an area near Quintard Mall, but, again, I could be wrong, and why "F", I have no clue.
Q. This map is entitled "Zones and Drainage Area for Remediation".
A. Yes.
Q. And again, I have asked you this once before; do you have any knowledge of

the basis for these zones?
A. Well, I mean -- the basis for the overall map is drainage. I mean, our plant sits in this big blank area to the lower -- as I'm facing it -- lower left-hand corner of the map. Our plant area is here (indicating) so that on the right side of the map, Snow Creek runs in a generally vertical manner going to the left. On the upper portion of the map, is an area that is the Ninth Street's ditch and the Eleventh Street's ditch which are drainage areas from our plant, and then an area from the drainage from the west end landfill, which is primarily the Ninth Street ditch, so that provided the focus for these areas because that's where EPA and Solutia had found PCBs primarily.

So it was basically here's the drainage pathway, we're going to move away some direction or some amount, and you can see it varies depending on where

people lived and where our plant was, we're going to move away from those drainage areas some distance not defined and label them as these areas and that's how it was generated to my understanding.

Q. That's what I assumed, but I wanted --

A. It's clearly drainage pathway driven.

Q. And could be what some people may call the flood plain?

A. Well, it's much larger than the flood plain. There are maps of the flood plain and they're certainly included in here, but this map is much larger than the flood plain.

Q. When you say much larger, can you quantify as far as feet or yards?

A. Well, it depends. Some places, it's very narrow and some places, it's very wide depending on what the flood plain looks like and whether the streams are constricted or not in those areas, so I

really can't generalize.
Q. Under the consent decree, the proposed consent decree, would all properties in these zones be tested?
A. Probably not each property. Going back to what we discussed earlier, that as we gather more and more information by our sampling, it is possible, statistically, to make some predictions about whether PCBs are likely or not likely to be in a given area and, subject to EPA's approval, we can terminate sampling in a given zone if we can statistically demonstrate to the EPA's satisfaction that we're not likely to find PCBs in those yards.
Q. But as you start the process, let's say you start in Zone 1 --
A. I'm not sure which one was first. Two and three were clearly the highest priority.
Q. So not No. 1?
A. No. These are not labeled by

priority.
Q. So two and three is considered high priority?
A. I believe. I think that's what it was. It says it in the consent decree specifically.
Q. Would you necessarily test every piece of property in zone two and three?
A. No, you would test every piece of property nearest the drainage things, moving away, and if you got to the point to where we and the agencies were comfortable that we weren't finding PCBs anymore, we could terminate even in those high priority zones.

Now, I believe, and, again, I'm moving a little further than I should, I think we've only terminated samples in one zone and I don't remember which one it is. Otherwise, we have not been able to demonstrate that is my recollection.
Q. You don't know if it's No. 1 or --
A. I believe it's No. 3, but I don't

know. I'm speculating. I shouldn't even be talking about this.

Q. What documents or what would I look to to see that?

A. There would be correspondence between us and the EPA.

Q. Okay. And as far as Zone 1, has there been testing been done to it?

A. I'm sure there has. I don't know.

Q. Again, that may be Mr. Branchfield?

A. Clearly. Clearly, Mr. Branchfield.

Q. Here's my question now: assume that this Exhibit No. 1 is, in fact, the map or assume it's the next map, whatever.

A. Yes.

Q. It's my understanding, then, if I have clients outside these zones, their property would not be tested for PCBs under the consent decree?

A. That would by my understanding, yes.

Q. And whatever properties are tested and the soil is found to have one part per billion --

A. Million.
Q. Million, excuse me.
A. With an "M".
Q. -- with an "M", be cleaned up?
A. We would make the offer to clean it up.
Q. And what would be the procedure for cleaning it up?
A. Removal of, basically, the top one foot of soil from the property and testing to be sure that some level isn't present underneath that one foot and then the one foot would be replaced with clean soil and revegetated.
Q. So if you clean the one foot off and then you test it again and still have one part --
A. No, I think it's ten at the one-foot level. I think it's ten.
Q. Okay. It goes to ten?
A. It goes to something else. I think it's a ten and that's consistent with EPA's spill cleanup policy for

residential areas which has been in place for a number of years.

Q. Have you known -- have you known or has it been known or has there been findings, I guess, of higher levels of PCBs than one part per million outside the zones that we're looking at here in Exhibit No. 1?

A. I don't know specifically. I do not know.

Q. You don't know one way or the other?

A. I don't know. I don't have that kind of detailed knowledge.

Q. Have you seen any type of a map that shows the various samplings that have been done in the Anniston area and the levels of PCBs found?

A. Yes, I believe I have seen such a map.

Q. And you don't recall any of those maps showing PCB levels being shown outside the zones?

A. I don't know whether I have seen it

superimposed on these zones or not.
Q. Okay. The Tox Profile we talked about earlier, I'm sure you're familiar with?
A. Yes.
Q. And I assume you know -- is it Dr. Copland?
A. I mean I know who he is. I've seen him. I've heard him speak. He certainly wouldn't know me. Oh, he's written me a letter so he may know me.
Q. Tell me, what is, in fact, the Tox Profile? What is it for?
A. Under the terms of the act creating ATSDR, the Agency for Tox Substance and Disease Registry, one of the things which is actually funded by EPA's superfund money, one of the things they were tasked to do was prioritize chemicals that were found at superfund sites.
Q. Okay.
A. So they have got lists of how many superfund sites benzene was found or

asbestos was found or PCBs were found, okay. That lists exists.

Q. I think I have that somewhere.

A. Yes, and it's almost universally misinterpreted. Okay, based on that list, EPA or ATSDR is then commissioned to either do or hire contractors to do, which is what they always do, an expensive literature review of everything that is known or everything that is found in the literature about, primarily, the toxicity of that particular compound, so there are dozens if not hundreds of toxicological profiles for a wide variety of chemicals, so that's what they are. This is one in a huge series.

Q. And --

A. And they are periodically revised. I think this is the third revision of the PCB Tox Profile.

Q. Is this the current --

A. It's the most recent, yes.

Q. And is this, and I think I asked you

this earlier, it says it's peer reviewed.

A. It is peer reviewed in the sense that ATSDR contracted with some number -- generally, it's like three scientists to review it and make comments.

Typical peer review, in the sense that it's usually meant, is an anonymous process whereby an absterer a draft paper is sent by a journal to, as I said, anonymous scientists with knowledge in the field to the review and make comments and those are then supplied to the author to make adjustments as they would. So this is not an anonymous peer review, but three scientists or so, in this case, many more did, in fact, review this document.

Q. Okay. There are certain parts of this you agree with and certain parts you don't agree with, I assume?

A. Yes. I'll say yes for now and see where you go with it.

Q. The reason I asked, is this an authoritative document for people such as you to look at to rely on as an authoritative?
A. To the extent that the document is a compilation of literature information on toxicity, environmental behavior, environment levels, a number of things out of the literature that were reviewed incorporated, yes, I would agree that it is an authoritative document and is relied upon and I rely upon it to the extent that interpretations based on that literature are superimposed on the factual basis of the document. I have some problems with the way things, some of the things that are said, but usually, more the way they are said.
Q. Okay, well let me just ask you, it its says here under Summary of Health Effects, Chapter Two, "Health effects that have been associated with PCBs in humans and/or animals include liver,

thyroid, dermal, ocular, immunological, alterations in neurodevelopment, mental changes, reduced birthrate, reproductive toxicity, and cancer." Is that one you would not agree with?

A. No, I will agree with it because it says humans or animals and that's the problem with that statement. Let's separate it out.

What do they say in humans? Now, I might not agree with that, but I agree that in humans or animals, I think most of those things -- I don't know about every single one of them, but I think most of them have been associated in some study or another.

And again, I think that illustrates my problem with the document in that it lacks for clarity in some instances.

Q. Okay. Let's take about a five-minute break.

(Whereupon, a short recess was

taken at this time.)

Q. I think I've gone over this, Dr. Kaley. Let me ask you one thing. This is the letter or, excuse me, the health consult of February of 2000 and I think we've talked about this, but on page six my copy which is, you're right, it's off the internet, so it's going to be different, but when they talk about the persistency of PCBs and the half lives of PCBs and they talk about the fact that if one -- let's see, talking about the range of half lives being from what?

A. Three to twenty four.

Q. Three to twenty-four, and basically say that if one has a level today, their level fifteen years ago would be twice as high or something like that. You don't agree with that statement?

A. No.

Q. I think you voiced that in your letter; correct?

A. Well, I think the most important

thing in this paragraph is the chain of ifs.

Q. Okay.

A. I mean, if you assume everything they're assuming here is correct, then I think their chain is not necessarily -- doesn't come to the wrong answer with all those assumptions, but I think I would have arguments, possibly, about every one of those ifs, you know, I mean, if exposures did happen years ago.

Well, maybe they did and make they didn't. We don't know. And I think somebody that has a very low level today, we don't know whether that came from today, ten years ago, twenty years ago, thirty years ago. It's very low. It's below or right at background anyway. To extrapolate that back just doesn't make sense, so I mean that's an if.

The estimated biological half lives for total PCBs; well, we've talked about -- that's total PCBs. We talked

about all the uncertainties in that. The three to twenty-four years, it's a big difference whether they use three years or twenty-four years on the half life.

And then I don't know what reference fourteen is. You know, I would have to look and see who did that work. But then they say, I mean, they are making -- if the elevated levels were caused by exposures that happened twenty years ago, you know, then.

They have set up a construct here which I don't argue with, you know, if you agree with all their assumptions, you may get to the point they do, but I don't -- there's all sorts of uncertainties around those assumptions, so I don't think you can take this paragraph to say that you can automatically do it.

Q. And so you don't agree -- from what you just said, I assume you don't agree, and I can't cite who says this, but that

levels of PCBs found in the general population of Anniston, Alabama is much higher than the general population found throughout the country?

A. No, I think the levels in some individuals are higher than levels found in other environmentally exposed populations, but I don't think you can make a general statement about the population of Anniston, Alabama.

Most of the people that have been looked at in the course of this litigation which is, I don't know what percentage of the people of Anniston it is, but whatever it is, most of those have been nondetects.

Q. Meaning, again, nondetect meaning what?

A. Meaning nondetect in terms of what the laboratory could detect or not. I mean, less than three parts per billion, less than five parts per billion, whatever it was, and another significant

proportion of what I call background range, so I don't think you can make statements about the whole population of Anniston, Alabama.

There are individuals who have levels that are higher that have been reported in background levels in other studies, but I don't think you can make a generalization about the whole population.

Q. Okay. Now, in this letter or this health consultation, they talk, and I think you referred to this on the soil sampling and dust sampling. In table nine, they talk about the samples that are were taken in various locations, and I want to you ask you do you know exactly where or do you know generally where those soil samples were taken?

A. Well, I mean, I may have at one time. I don't right now. As I'm sitting here, I don't even -- oh, community group one. Okay, I was going to say I didn't

even know what CG-1 and CG-2 are. That's community group one and community group two. No, I don't know -- I mean, obviously, there's six hundred fifty-five samples from community group one. Those are all plaintiff properties from Mr. Stewart's clients. I don't know where each one of those are.

Q. And it mentions some of those were taken outside of the flood plain?

A. Yes. For instance, community group one, it says in flood plain connected to Solutia and for GC-1 it says mostly no.

Q. And were those in CG-1, that testing in CG-1 outside quote "the flood plain" where they found -- did they find high levels of PCBs?

A. You can't tell from this graph and I don't know without looking at individual properties.

Q. But again, the position Solutia takes, if in fact, that did occur; that is, if high levels were found, it got

there some other way other than through the drainage ditches, etcetera?

A. Yes.

Q. I.e., field dirt for instance?

A. Yes.

Q. Is that the main source of that?

A. In my opinion, as far as I know, yes. Now, I would also add that that doesn't mean we're not cleaning them up if they're in the areas that we've agreed to clean up properties. We're still cleaning them up.

Q. Like the OLN, the Oxford Lake Neighborhood, for instance?

A. Right.

Q. For instance, CG-1, the six hundred fifty-five samples outside, most of them being outside of what the call the flood plain?

A. Right.

Q. The maximum was eight hundred forty part per million?

A. Yes.

Q. That's pretty high, is it not?
A. For a residential property, yes, I would admit that's elevated.
Q. And in the range of the top ten, I'm not sure I understand the range, seventeen point four to eight hundred forty?
A. Well, if you rank them one through six hundred fifty-five, number one was eight hundred forty and number ten was seventeen point four and they went down from there.
Q. Okay.
A. That's the range of the top ten samples, so obviously, it drops real fast.
Q. Okay.
A. But I don't whether that eight hundred forty or the seventeen was in or out of the flood plain. That's why I can't answer your question. I don't know where those samples were. Those top ten may have very well been all in the flood

plain.
Q. You don't remember that?
A. I don't know if -- no, I doubt if I did know at one time.
Q. Now, I recall seeing something and, again, it may have been a newspaper article, I can't tell you, but, something about Solutia and/or Monsanto using mercury in their processing?
A. Yes.
Q. What exactly would that use mercury for?
A. Mercury was used in the process -- we made chlorine at the Anniston plant from the mid-1950s, I want to say 1955 to 1969 and the process we used is an electrical chemical process and mercury was used as one of the electrodes in that process.
Q. Did you say '55?
A. Yes.
Q. To '69?
A. I believe it's that general time

frame. I know the '69 is right. I'm not so sure, it could have been '54 or something like that.

Q. And was this mercury used on a closed loop basis?

A. Yes. That was certainly the intent.

Q. Meaning it was recirculated or recycled or whatever?

A. Yes.

Q. And was there any determination made at any time that some of this mercury was being lost in the environment?

A. Yes, there were discussions, certainly, efforts to control that from happening which would suggest that there were some amounts being lost in the environment, yes.

Q. And when were those efforts made?

A. In the mid- to late-60s primarily.

Q. And was that successful?

A. To a large extent, yes.

Q. What do you mean?

A. Well, you can't ever get it -- there

are parts -- some of the kinds of mercury that ended up being in discharge waters are much more difficult to control than others so if the elemental mercury, the little shiny silvery stuff we're all familiar with, which is what we used primarily, that's fairly easy to control. You can separate it out. You can see it. But once -- some of it reacts to soluble water, soluble forms of mercury, and then those are much more difficult to control although efforts were made.

Q. Has there been some heated debate, if you will, about whether or not Solutia admitted that there had been some discharged into the drainage ditches of Anniston?

A. I do not believe there's heated debate about whether. I think, certainly, there was an article in the Anniston Star and I responded to that article and I think that the levels, the

numbers they were reporting in that article were overestimates of any mercury that could have been lost to the environment.

Q. What was their estimates; do you recall?

A. I don't remember. It was very large.

Q. And you responded to them by saying there was no way that it was that large?

A. Yes.

Q. Was there an effort made by Solutia or Monsanto to determine what quantitative amount was being lost?

A. I did an extensive review of the available documentation and my conclusion was that it was not possible to make that determination, but also pointing out that it, in some ways, didn't matter what it was because we and the EPA have looked for mercury in the drainage pathways and residential yards, to some extent, and in the waterways and mercury is not there at

elevated levels.

Q. Well, as I understand, there were attempts, at least in the seventies, or the late-sixties, I guess, to determine what level of PCBs were escaping?

A. My recollection of my document review was that there were attempts to limit losses of mercury in the process, but many of those losses were on-site losses and that's where I think the difficulty -- somebody took a number that was including on-site and off-site losses and projected what that would be if they were all off-site losses and that's how that huge number got generated.

The documents are clearly -- many of those losses were losses that were on-site or materials that were recovered and later put back in the process and things like that, so there were clearly efforts to look at where the process is mercury was lost and what can we do to stop it and, you know, part of

that is what can we do to keep it from get in the environment, but a lot of it was what can we do to control it in our process.

Q. When you say on-site loss, how would that --

A. Spill on the ground. Leaky -- they were in these big cells. The cells leaked to some extent, things like that. Things that we can do to, you know, recover things better. I don't remember exactly what they all were.

Q. Do you recall, in analyzing the documents determining how much was spent on mercury on an annual basis?

A. I think that number is actually in there. I don't know what it was, but it was a significant amount of money and, frankly, there was an economic impotence to recover the mercury because it was expensive to replace. I believe that number exists. I don't remember what it was.

Q. What about the loss or was there any lead used in the process?
A. For a short period of time, there was lead used in the process to make biphenyl which is the starting material for PCBs. It was basically done in lead pots.
Q. And in what period of time?
A. I should know, but I don't remember. Probably started -- it was probably from early on and I think we started changing those out in the mid-fifties, but as I'm sitting here today, I don't remember the dates on that.
Q. Do you remember whether or not there was an effort made to prevent the escape of the lead into the environment?
A. Yes. Well, part of it -- I mean it was called a lead pot process and there was really very little chance for lead to escape in the environment anyway, but, again, my recollection of my review was

that there was essentially no chance for lead to get out of that process from our facility.

Q. So there was no lead escaped as far as you know?

A. Not at measurable levels, I don't believe.

Q. Has that been questioned in the recent past?

A. Not really. I think -- I don't believe so.

Q. Well, I didn't know if that particular article, and I don't remember the article myself, but whether it addressed mercury and lead or just mercury?

A. Well, I don't remember either. There was one on -- I think there was at least one article on both and then your question tweaked one of the -- I think there was a recent article on one of them, kind of a review of where it was, but I thought that was the mercury one.

Well, it may have been the lead one actually, because of the lead site health consultation that was released so it may have been the lead. I don't remember. I mean, the articles are obviously out there to be checked, but I don't think anybody's questioned what we're saying about our use of lead in the facility.

Q. No, I'm talking about the escape of the --

A. I don't think anybody is even questioning that, whether significant levels had escaped. I'm not aware of that, and, again, we have done lead sampling around our site and really have not found any evidence that the site is contaminated with lead.

Q. Now, I'm going to switch back over here to the AST -- the Toxic Profile on the area of cancer that we've both talked about, I think, extensively today, but let me just ask you a couple of things

about that profile.

"Carcinogenesity of PCBs in humans has been investigated in retrospective occupational studies that evaluating cancer mortality in worker's exposure in capacity, manufacturing, and repairing," and I think that refers to the Bertazzi and the Brown and the other studies, I assume?

A. Yes.

Q. "And in case control studies of the general population, they examined associations between cancer and serum or out of those tissue levels of PCBs based on indications of PCB-related cancer at several sites, particularly the liver, bilary tract, intestines and skin, melanoma, the human study provides suggestive evidence that PCBs are carcinogenic."

Would you agree with that statement that it's suggestive evidence?

A. I would agree with that in the terms

that suggestive is used and those agencies that rate carcinogenicity, that it probably meets the criteria for that.

Q. Well, let me talk about that, and maybe I don't understand what you guys mean by suggestive because I made comments earlier that, you know, law and medicine don't mix and I guess it's the same way with science and law don't mix, so what does suggestive mean in the scientific field?

A. Well, primarily, the term "suggestive" comes out of IARC, capital I-R-A-C, you why which is the International Agency for Research on Cancer, and like the EPA, IRAC has a carcinogenicity rating scheme for chemicals and their scheme is based on two and maybe two and half criteria.

The first criteria is animal carcinogenicty and if something is an animal carcinogen, it automatically becomes a probable human carcinogen in

that scheme, so it doesn't matter if we have any human data at all.

Q. Okay.

A. Then they look at the human data and if there's any report of any association they typically call that suggestive. So they look at it and they say it's inadequate, it's suggestive, or it's conclusive, all right.

So the people on that committee, one of which -- the committee was headed by Bertazzi who has written one of the studies, uncoincidentally enough, made a determination that based on Bertazzi's study and others, that there were enough associations reported in that literature to call it suggestive. But it certainly did not rise to the level of conclusive which would have made it a known human carcinogen.

Q. And when you say a know or conclusive is that -- can you quantify --

is that ninety nine percent?
A. No. I don't know what it is. In IRAC's assessment, in general, it means that -- they just basically put together a group of scientists and that group of scientists, some of them look at the animal data and some of them look at the human data and if the group looking at the human data does a weight of the evidence assessment concludes in their minds that there's enough human data on that compound, there are enough positive studies, there's consistency, there's strength of association, all those Bradford Hill criteria, I'm sure you have heard about until your ears are full, then they will make that determination.
Q. That it is --
A. That it is a known human -- that the human information is conclusive. I think the term they actually use is "adequate", and that it is a known human carcinogen.
Now, we have talked about

dioxin, and they bent their rules on dioxin because the human epidemiology group decided that there was not enough information on dioxin to call it a known human carcinogen, but the overall committee -- I'll save my rhetorical or editorial comments -- the overall committee decided that there was enough known about the mechanism of the cancer in animals and the fact that that mechanism probably occurred in humans that they were going to name it a known human carcinogen even in the face of inadequate information, so they kind of tweaked the --

Q. What is the difference between probable suggestive and conclusive? Is conclusive synonymous with known carcinogen?

A. If the animal and human evidence are conclusive then it becomes a known human carcinogen under their classification scheme.

Q. And if it's suggestive, it becomes probable?
A. It remains probable.
Q. Okay. So you may have animal studies that show --
A. Are conclusive.
Q. Are conclusive, so therefore, it's a probable causation?
A. Yes, automatically.
Q. If you have human data, it becomes suggestive?
A. It can be. I mean, you can have inadequate data which says there's really nothing there or there could be negative human data which would probably still leave it a probable or you could have what they call the suggestive which is, again, in the interpretation, I'm not sure that I would agree with their interpretation even under suggestive.

I think there's just too much inconsistency in those studies. But that was what that group said and I'm not

going to, you know, there are positive associations in some studies. I'm not going to argue with that.

Q. Would you agree that suggestive as used by this group means that it's more probable than not that it's carcinogenic?

A. No.

Q. Would it be less likely than not that it's carcinogenic?

A. Well, you're asking me to get into the minds of a group of I don't know how many people that were meeting in Leon, France fifteen years ago. I think it means it's less probable than not, but that's based largely on my view of the human literature and not so much on what I think they were really thinking.

Q. Well, when they say probable human carcinogens --

A. But we're getting back. That's just what they call it because of the animal. I mean, you don't have to have any human data for it to be a probable human

carcinogen. It's just based on the fact that we think we know enough about this chemical, and I don't even care whether it's PCBs or what it is, about this chemical based on animal studies that, as regulators and people chartered to be protective of human health in the overabundance or abundance of caution, we're going to put this label on it which says you need to think about this possibility in your research and in your regulation of this material. It doesn't mean that it has ever caused a cancer in a single person.

Q. But PCBs are classified as probable carcinogens; is that correct?

A. They're classified as probable human carcinogens, that is correct.

Q. And by whom?

A. By IARC and the US EPA.

Q. And IARC is who?

A. International Agency for Research on Cancer.

Q. And who exactly is that?
A. It is what it is. I think it's associated with the World Health Organization, WHO, and it's basically a group that that's what they do is they get together and talk about the information available on candem. The primary purpose is to say okay, we've got "X" resources we can spend on cancer research, how are they best spent, well, let's not waste them on things that are non-carcinogens and maybe let's not waste them on things that are known carcinogens because we already know that, let's think about what research could we do on something that we need to learn more about. That's really the kind of purpose of their classification is to guide research into those areas.
Q. Does that same agency classify dioxins as known carcinogens?
A. IARC classifies dioxins as known carcinogens. I just talked about the

process by which they did that.
Q. And as does EPA?
A. EPA is trying to do that. They have not. That is one of the controversial proposals of their still draft dioxin reassessment.
Q. And as I understand from this afternoon's discussion with you, you don't agree that dioxins even are a known carcinogen?
A. I don't agree that the human evidence for dioxin carcinogenicity in humans is sufficient to classify it as known.
Q. So you don't agree with IARC; correct?
A. I don't agree that dioxin causes cancer in people.
Q. And you don't agree with IARC as it relates to PCB being a probable carcinogen or do you?
A. I agree that it meets their classification for probable carcinogen. I

don't think it means it probably causes cancer in humans. And I think there is a distinction there. I do not argue that it meets their criteria or EPA's criteria, for that matter, as a probable human carcinogen because it is positive in animals.

Q. And it's only positive in animals?

A. Pardon?

Q. And it's only positive in animals?

A. It's positive in only animals.

Q. That's the distinction?

A. Yes.

Q. I probably should have asked you this before we started, but have you reviewed any other matter, specifically in preparation for this deposition or this particular case, the Tolbert case?

A. Well, with regard to the Tolbert case, in general, no. I mean, I've looked at the expert reports, I guess, on both sides.

Q. Okay.

A. And I've read a couple of depositions so for Tolbert, that's what I've done.

For this deposition specifically, I reviewed my -- I have an affadavit, kind of a historical summary affadavit that has been filed in a number of cases. I did review that.

Q. Okay. But as far as any data, I know you say you've looked at the expert reports, but have you look at any specific data that might be associated with those experts reports? I think you said, generally, earlier that you --

A. I've looked at the blood data and I've looked at Hanson's calculations and stuff like that.

Q. Is there anything specific about the data, the blood samples and/or the soil samples that you that jumps out at you as being incorrect or do you have any criticisms about it as you did in the letter to the ATSDR?

A. I think there are some obvious inconsistencies between the data generated in the Tolbert case, the data generated by, I think it's Acculab, that the total PCBs and the Access Lab that did the individual congener PCBs and part of that is because I'm not familiar with Access Laboratories and their capabilities to do what I know to be a very difficult analysis so it's hard somehow to rationalize those differences. Some of there are explanable. Some of them aren't. I've thought about it, but I really haven't reached any conclusions.

Q. So there's some discrepancies, you say, in Accucam's levels that they found and the total congener or total congeners at least analyzed by Access? Of course, there's going to be differences. I think there are going to be -- strike that.

If you take the two hundred nine total congeners and analyze those,

aren't you going to get a higher number as opposed to taking only ten and analyzing those?

A. It depends on which ten.

Q. Correct.

A. And it will be somewhat higher, but not necessarily a whole lot higher because most studies show that about sixty to seventy percent of the human body burden is accountable for about four or five congeners so I would say, you know, you may get a factor of one and a half or two higher just because you're doing all two hundred nine, but you wouldn't expect it to be much more than that.

Q. And is that the discrepancy you're talking about; that is, that the factor is maybe three times higher?

A. Yes, and I don't remember the data, but there are some that are higher. I mean, some of them are whole blood and some of therm are serum. That's going be

a factor to be a difference, so I think there's some reconciliation that needs to be thought about in some of those data. I don't have any basic objections that one is right and one is wrong, here's why it's right and here's why it's wrong.

Q. Okay. You just observe some discrepancies --

A. And there's going to be. I mean, I think we talked a little before, too, that the science or art of doing analytical chemistry with PCBs is very difficult. When you do that in human body tissues or any body tissues, animal or human, it is even more difficult because of the very complex matrices.

Blood a very complex, very difficult thing to work with to get all the PCBs out, you know, and get them analyzed and things, so there's going to be analytical variations superimposed on both of those laboratory's results.

One of the things I don't know

and haven't seen and maybe don't exist are the quantifications of how those laboratories do on a daily basis on a PCB analysis, how wide is their variation. It could be plus or minus twenty percent, it could be plus or minus one hundred percent on just daily variation in the lab.

And I haven't seen those kinds data. I don't know if they are even available to help make some of those decisions, but there's nothing in either set of data that I see that says throw it away, you can't rely on it for anything.

Q. You just need some explanations as to some of the --

A. Yes, I need to think about it more and I haven't.

Q. You'll think about it more between now and October?

A. Probably.

Q. In your letter, again to the ATSDR, an which is dated April 2000, you make

this comment: "It is notable that among the largest group of plaintiffs suing Solutia, not a single physician or other medical expert has opined that PCBs have caused a specific health condition in a specific individual."

You have read, have you not, the expert reports in this case?

A. Yes.

Q. Have you found that there has been, at least in this case, some physicians who have opined that certain plaintiffs exposed to PCBs do have adverse health conditions?

A. Yes, I'm aware of that, but at the time, you weren't the largest group.

Q. Okay. We weren't even a group.

A. That's right. That was not addressed to your group of plaintiffs.

Q. Okay. Have you had any conversation with any physicians concerning the health effects of either this plaintiffs' group or any other plaintiffs' group that you

can recall?
A. That's a very broad question. I believe that some of our experts are physicians and with some of our experts, I have probably had some discussion about the health of this plaintiff group or others, but I don't have any specific recollections of that. I mean, is that what you meant?
Q. Well, as the director of environmental affairs for Solutia, is it part of your job to analyze or determine, if you will, the health effects or possible health effects on the community from PCBs?
A. I would say that's part of my responsibility, yes.
Q. And as part of that responsibility to do that, do you, on a regular or frequent or other basis, consult with physicians to keep abreast of what, if any, health effects there might be associated with exposure to PCBs?

A. No, I do not. I rely primarily on the literature or discussions with other scientists, but some of those may have been physicians, but I don't do it routinely.

Q. And when you say you rely on the literature, do you -- you obviously have some or you obviously don't take the literature that we discussed in a general nature and come to the conclusion that PCBs, for instance, causes cancer in humans?

A. I have not come to that conclusion based on my review of the literature, that's correct.

Q. But you have seen reports of various scientists, if you will, that do come to the conclusion that those studies indicate or suggest that there are cancers associated with PCBs?

A. Well, I mean, I guess we could get into a conversation about the difference between causation and association if you

want to do that, and are you talking about in the literature or are you talking about in expert reports?

Q. Well, the literature first?

A. Certainly, there is literature which says that PCBs are associated with various cancers and various individual studies. I think we had that discussion. I don't think any one of those pieces of literature says PCBs cause cancer in humans.

Q. So as I understand, you would need to have a study or studies that will conclusively show that there is, in fact, a conclusion that PCBs causes cancer?

A. I would say for me to reach that conclusion, it would take a robust set of studies of some size which show strong associations, consistent findings of specific cancers and in specific people. I mean, the whole Bradford Hill criteria thing.

I'm sure you get tired of

hearing about them or maybe you don't, but those criteria are there for a very good reason and they are accepted by -- widely within the epidemiological community and I believe that that is because they make sense and they provide, although there is judgments within the various things, they provide a relatively objective set of criteria against which you can look at a body of literature.

Q. Now, all eight criteria don't have to be met do they?

A. All eight criteria have to be considered. I wouldn't say they have to be met, but certainly, there are some that are more important than others.

Q. So would you need to have an epidemiology study as such or just --

A. I don't think any epidemiology study necessary tells you anything. You need a body of literature upon which you can rely.

Q. And if that were to happen, if you

had a quote "robust body of literature" that confirmed that cancer was caused by PCBs, would there be -- what, if anything, would Solutia do if that were to occur that they're not doing now?

A. I don't know specifically what we would do. We will certainly change our public pronouncements on our view of the carcinogenicity of PCBs in humans.

Q. Do you know whether or not Pharmacia has a different policy as to whether or not PCBs is carcinogenic, causes cancer in humans?

A. I have no idea.

Q. Has that ever been discussed with Pharmacia?

A. Not by me or with me present.

Q. And I assume you have not had any or have you had any conversation with anyone with Pharmacia about PCBs?

A. I don't believe so. Well, not in the context of Pharmacia. There are people now with Pharmacia who I may have

had that conversation, but that was when we were all happily Monsanto and I don't have any specifics, but I don't want to say it was nobody, because it may very well have been.

Q. Was there anyone that would be your counterpart with Pharmacia that was with Monsanto?

A. Not that I'm aware of.

Q. Was there anyone that left Solutia to go to Pharmacia that might be an environmental affairs person?

A. I'm sorry?

Q. Has anyone left Solutia --

A. Has anyone left Solutia to go to Pharmacia?

Q. Yes.

A. I don't believe so, not that I know of.

Q. I think you mentioned the dioxin reassessment.

A. Yes.

Q. What exactly is that?

A. A very long story. I'll be brief. It is an attempt by the EPA to do a comprehensive reassessment of all the toxicological information and human health information known about, primarily, TCDD, but encompassing the broader category of what we've jokingly been calling dioxin-like compounds in and effort to provide information for risk assessors and risk managers about what to do with dioxin contaminated cleanup sites, primarily, but also having implications for dioxin levels in the human population.

Q. And it addresses the areas of the dioxin-like PCBs?

A. Yes, it does. Briefly, but, yes, it does.

Q. And does it conclude that the coplanars or dioxin-like PCBs are similar to the dioxins or the TCDD that causes cancer?

A. Okay, I'm going to jump to where I

think you were going --

Q. Okay, thank you.

A. -- and say does it conclude that dioxin-like PCBs are known human carcinogens. The answer to that is no, it doesn't even make at that determination for the other dioxins or the dibenzofurans, nor did IARC.

The designation of dioxin as a known human carcinogen is the single compound 2378 tetrachlorodibenzodioxin, what we've been calling TCDD, it does not address the carcinogenicity of any of those other compounds and, in fact, IARC specifically declined to address the carcinogenicity of those other compounds excluding the PCBs. The PCBs weren't even in the IARC's discussion.

Q. The EPA's proposed reclassification of TCDD, as I recall and I may have it, to human carcinogen mixtures including dioxin-like PCBs congeners are quote "strong cancer promotors in weak,

direct, or indirect initiators and are likely to present a cancer hazard to humans." Is that contained in that --

A. I don't know. It would not surprise me if that is a statement take out of the dioxin reassessment.

Q. Why?

A. Well, because that's the view the EPA is trying to profess.

Q. Again, you don't agree with that?

A. I do not agree that the data available from human or animal literature support that conclusion. No, I don't agree with that and nor did a number of members of the advisory board that reviewed the dioxin reassessment, by the way.

Q. They got outvoted?

A. No, it's still up for discussion. That's why it's still a draft document and that's one of the main reasons why it is probably going to the National Academy of Sciences for another assessment before

it's ever released. That's one of the primary discussions.

Q. And who are the members of that --

A. That has not been established. The committee has not been established. There is merely a proposal that it be reviewed by the National Academy of Sciences.

Q. All right, now you do agree, I think, that PCB exposure does, in fact -- let me make sure I get the words right for you, an elevation in liver enzymes; is that correct?

A. High levels of PCBs -- there are reports of high levels of PCBs causing transient elevations in some liver enzymes, yes.

Q. But as I recall, initially, today, you said that there is no indication that that is necessarily a risk?

A. Well, I mean, it indicates that the liver is somehow reacting to that exposure to PCBs. I think what I said

was that that has not been demonstrated in the literature to be followed by frank liver disease.

Q. Okay. So are there studies which support the Hill criteria of strength of the association between the observed effect to the PCBs and liver damage? Is there a strong association between exposure of PCBs and liver damage?

A. No, I do not believe there is.

Q. Are you familiar with Maroni?

A. Yes.

Q. And is it Fishbein?

A. Fishbein.

Q. Do those studies not indicate --

A. I don't remember. They may, but there are only two. I mean, it goes back to the other ones. There's two among twenty or thirty studies that have looked at that end point.

Q. Not robust enough is your --

A. Well, not consistent enough and frankly I don't remember Fishbein talking

about frank liver damage. I think Maroni may have been looking at porphyria, but I don't think that was followed up in a second study. I don't remember Fishbein doing it. I could be wrong, but I don't recall that.

Q. And I don't have it with me, so I don't know either.

A. I think his was one of the ones that talked about liver enzyme elevations.

Q. Then you do not agree that high levels of PCB exposure will cause liver damage?

A. They will in animals for sure. I do not believe human populations have been exposed -- I do not believe that human populations have been exposed to levels that would cause that if they would.

Q. Okay.

A. I'm not going to say there's not some level of -- you couldn't force enough PCBs down my throat that my liver wouldn't be damaged at some point. I

think populations exposed, as workers are everywhere, we haven't seen evidence of that actually coming to the fore.

Q. In other words, if you consider the general exposure levels in the general population, which you mentioned, I believe earlier, you don't believe that that is a level sufficient to cause liver damage?

A. That's absolutely correct because you don't see it in the more highly exposed occupational studies.

Q. Have you ever had your blood serum tested for PCBs?

A. No, I haven't.

Q. Have you ever had a liver profile done?

A. I have liver profiles done in my annual physical.

Q. Is there any elevation in your liver enzymes that you know of?

A. Not that I know of.

Q. Do you know of anyone that works for

Solutia that has had their serum level for PCBs performed?

A. Yes.

Q. Okay. Who?

A. I know that it's number of them.

Q. Is that something that has been routinely done?

A. No.

Q. Is it something that used to be routinely done by Monsanto for their workers?

A. No.

Q. It seems like I've seen something in the documents that suggested that. Am I wrong? Do you remember seeing some documentation or memos concerning having periodic testing done for -- I misspoke. Is there anything in the literature that suggests that certain workers have their liver profile performed after being exposed to PCBs?

A. Monsanto workers or workers --

Q. Monsanto workers?

A. Oh, I don't know. I'm not aware of anything.
Q. I think my question seemed to indicate I was talking about the blood serum, but what I meant to say was their liver enzymes.
A. I'm not aware of that either.
Q. Okay. Since the Abernathy trial or since you testified in the Abernathy trial -- it's still going -- have you given your testimony in any other case, PCB-related case?
A. No.
Q. And I believe you testified in March about year ago; is that your recollection?
A. That's the latest time, yes. I mean, I testified in the Gadsden phase and the environmental -- what's the word I'm looking for -- injunctive relief phase, so I know two instances of my testimony in that litigation. I believe this is my first deposition in a case

since that time.
Q. Are there still ongoing communications with the EPA and yourself? Is there something going on that y'all routinely communicate?
A. Not me. Now, Craig does. I mean -- well, let me say we are continuing to submit documents in anticipation of the eventual entering of the consent decree and I review those documents, but there is nothing going to the EPA under my name.
Q. What about the Alabama Department of Environmental Management, have you had constant contact with them?
A. Not recently, no.
Q. There's a letter here dated -- I don't know where I got it.
A. Wait, stop, I do, because we have to report on a bimonthly basis air levels from our air monitoring program to ADEM and EPA and I write those letters, so yes.

Q. March 10th of 2003; is that the most recent letter?
A. That's the most recent.
Q. Okay. What's the purpose of your having to write them?
A. Do you want a twelve-year-old kid's answer?
Q. A short answer.
A. They told us to. No, we have been doing air monitoring. We had proposed to stop air monitoring because we don't believe it is providing a whole lot useful information. They have requested that we continue to do that air monitoring and provide them with the results.

Now, starting last month or this month, we are entering into a more useful air monitoring program under the oversight of EPA and ADEM which will include meteorological data, and that work plan was approved and is being implemented, so the air monitoring will

go on. These letters, presumably, will cease at this point.

Q. But as far as the EPA is concerned, you have no continuing dialogue with them concerning the proposed order or the consent order?

A. Me, personally?

Q. Right.

A. That's correct.

Q. Is that something that will -- will you eventually do that or is that --

A. Probably not. I mean, that's largely Craig's responsibility is to work with a EPA and the implementation of that consent decree. I will continue to have an advisory role with Solutia and I very well may meet with EPA on a, you know, an ad hoc basis at one time or another but that is -- my daily or weekly or monthly interactions on a continuing basis is not part of my responsibility.

Q. So the implementation of the proposed order, if it's agreed upon or

ordered, it would be Branchfield?
A. Branchfield, yes.
Q. And did you have any, I guess, input into the actual drafting of the proposed order itself?
A. Some.
Q. But would he have more input than you?
A. Yes, but, by far, the EPA had a lot more input than any of us.
Q. And what do you see as your, other than a consulting role, what do you see as your role in implementing the proposed consent decree?
A. Other than advisory consulting, nothing. I mean, you know, I'll continue to review documents that we submit. I'll continue to review things coming the other direction, but other than that, nothing.
Q. Who will actually do the -- under the proposed consent decree, who will actually do the actual soil sampling and

testing?
A. Our contractor to Solutia. We hire people to do those things.
Q. Is there a particular one that you use?
A. There's one we have been using.
Q. Is that one you think you will continue using?
A. Probably.
Q. Who is that?
A. It's Genesis Project, Mike Price. I need to take two seconds or two minutes.
Q. Okay. Let's go off the record a minute.

(Whereupon, a brief recess was taken at this time.)

Q. With respect to the cleanup or assuming the soil samples indicate there's one part per million, who actually does the reclamation or remediation?
A. We've got us a contractor. I don't know -- I don't really know who's doing

that.
Q. So is that a bid-out job type situation?
A. I assume so. It was probably bid out the first time. After that, it's probably been the same person.
Q. Okay. All right, I think that's all. Thank you, sir.
A. You're welcome. Thank you. I appreciate your courtesy.

(The deposition of Robert G. Kaley, II, Volume I, was adjourned at approximately 5:15 p.m. on May 1, 2003.)

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REPORTER'S CERTIFICATE

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STATE OF ALABAMA

COUNTY OF JEFFERSON

I, Angela Abbott Blankenship, Certified Shorthand Reporter and Notary Public in and for the State of Alabama at Large, do hereby certify that on May 1st, 2003, pursuant to notice and stipulation on behalf of the Plaintiffs, I reported the deposition of ROBERT KALEY, II, who was first duly sworn by me to speak the truth, the whole truth, and nothing but the truth, in the matter of ANTONIA TOLBERT, et al., Plaintiffs, versus MONSANTO COMPANY, PHARAMACIA, INC. And SOLUTIA, INC., Defendants, Civil Action Number CV-01-C-1407-S, now pending in the United States District Court Northern District of Alabama, Southern Division, that the foregoing 208 typewritten pages contains a true and

accurate transcription of the examination of said witness by counsel for the parties set out herein; that the reading and signing of said deposition was not waived by the witness and counsel for the parties.

I further certify that I am neither of kin nor of counsel to the parties to said cause, nor in any manner interested in the results thereof.

This 12th day of May 2003.

_____

Angela Abbott Blankenship
Reporter and Notary Public
State of Alabama at Large

Transcript Word Index

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