

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.

Kaley, Robert; Tolbert

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.

* * * *

Kaley, Robert; Tolbert

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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

Kaley, Robert; Tolbert

1 BY MR. RODEN:

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

4 A. Robert George Kaley, II.

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

7 A. Director of Environmental Affairs.

8 Q. And you have held that since what
9 year?

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

15 Q. Okay. And where are you physically
16 located?

17 A. In St. Louis.

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

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

23 Q. Okay. In St. Louis?

1 A. Yes.

2 Q. Have you ever worked in any other
3 facility or been anywhere other than St.
4 Louis?

5 A. Not located there for any length of
6 time.

7 Q. Your office, in other words?

8 A. Correct. Exactly.

9 Q. And who do you report to?

10 A. I directly report to Tom Bistline,
11 B-i-s-t-l-i-n-e.

12 Q. What's his position?

13 A. He's an attorney.

14 Q. Is he general counsel or in-house
15 counsel?

16 A. He's assistant general counsel, I
17 believe.

18 Q. And who would work under you? What
19 titles are people who work under you?

20 A. I have -- as Monsanto employees
21 there's a manager of -- I don't know
22 whether he's -- I think he's manager of
23 environmental technical support and then

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

4 Q. What does she do?

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

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

9 A. Generally, yeah, a variety of
10 things.

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

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

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

20 A. No.

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

23 A. No.

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

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

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

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

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

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

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

15 A. Primarily Craig Branchfield.

16 Q. How do you spell that?

17 A. Branchfield?

18 Q. Yes.

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

20 Q. What's his position?

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

23 Q. And do you frequent the Solutia

1 facility in Anniston?

2 A. I don't know what "frequent" means.

3 Sometimes more, sometimes less. I'm

4 probably there on an average of once a

5 month.

6 Q. What exactly is the facility

7 manufacturing today?

8 A. It manufactures a chemical called

9 biphenyl which is a component of heat

10 transfer fluids and it manufactures a

11 chemical called para-nitrophenol

12 p-h-e-n-o-l, which is a raw material for

13 analgesics.

14 Q. Prior to that, when was it exactly

15 that Solutia or, I guess, really the

16 Monsanto facility ceased producing PCBs

17 in the Anniston plant?

18 A. 1971.

19 Q. Now, do you, as director of

20 environmental affairs, keep up with the

21 various samples, the soil samples that

22 are being conducted by various groups in

23 the Anniston area?

1 A. From a very high level viewpoint,
2 yes.

3 Q. What do you mean?

4 A. Well, I mean I see the results and I
5 think about what they look like and, you
6 know, overall and what they're telling
7 us, but I don't necessarily look at each
8 sample individually and try to determine
9 whether it makes sense with the one next
10 to it or things like that, but I have a
11 general overview, overknowledge of the
12 soil sampling results.

13 Q. Would that include Dr. Bonner's soil
14 sampling?

15 A. I am aware of those. I haven't
16 reviewed those in any great detail.

17 Q. Have you reviewed those in relation
18 to the Tord Case?

19 A. Not specifically.

20 Q. You have reviewed soil samples in
21 relation to the proposed settlement
22 agreement between EPA and Solutia?

23 A. Yes. Again, at a very high level,

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

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

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

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

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

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

6 Q. What is the current situation with
7 that sampling?

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

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

23 A. Well, basically, when the

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

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

18 A. No.

19 Q. Is there any radius involved at
20 all?

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

23 Q. Who decided the areas, the EPA?

1 A. Yes.

2 Q. Did y'all have any input into that?

3 A. Not really, no.

4 Q. Do the areas include properties
5 outside what is called the flood plain?

6 A. Yes.

7 Q. Is it any particular distance from
8 the flood plain?

9 A. Well, the distance is varied
10 depending on -- I mean it's not -- the
11 areas are not set by okay, here's the
12 flood plain and you have to go half a
13 mile past that or anything like that. I
14 really don't know what their -- there's
15 nothing, no rhyme or reason that -- I
16 have never looked for one, but I don't
17 see one obviously.

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

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

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

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

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

18 A. Yes.

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

1 anybody can answer that question.

2 A. Well, I'm not sure they can either.

3 I mean the first step, obviously, would

4 be to -- there is a map that exists.

5 Q. And I may have it somewhere.

6 A. I'm sure you do, because if you got

7 a copy of the consent order, I'm sure you

8 do have it, that lays out those

9 particular areas. The first step would

10 be to determine where those people lived

11 in priority -- within those areas.

12 If they live within those

13 areas, then they are certainly, more

14 likely than not, to be sampled. If they

15 are outside of those areas, under the

16 current consent order under which we're

17 operating, I think there's less

18 likelihood that they would be sampled.

19 Q. Okay. Do you know whether or not

20 the agreement to, or the proposal for

21 those areas was determined by any factors

22 that were used to determine that? Do you

23 have any knowledge of that?

1 A. Okay, now, let me try to clarify
2 something. You keep talking about a
3 proposal. I'm talking about the consent
4 order under which we are currently
5 operating.

6 Q. Okay.

7 A. Now, there is a proposed consent
8 decree which is a different animal, so
9 I'm talking, and I hope you are, about
10 the consent order under which we are
11 operating. And my understanding, as I
12 said, I don't really know how that was
13 conceived. But my understanding is it
14 was based on sampling that the EPA had
15 done in the Anniston area and roughly
16 encompassed those areas where, in their
17 sampling, they found some properties that
18 had PCBs on them.

19 Q. Now, what is the difference between
20 the order in which you are working and
21 the consent proposed as far as the area
22 is concerned that would be sampled?

23 A. Well, as far as I know, and I'm not

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

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

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

17 Q. But the areas would still be the
18 same?

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

1 identified that were impacted by PCBs.

2 Those would be sampled.

3 Also, the consent decree, the
4 current consent orders address only
5 residential properties. The consent
6 decree would address other kinds of
7 properties; public access properties,
8 things like that.

9 Q. Schools?

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

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

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

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

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

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

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

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

21 Q. What do you mean by streamline risk
22 assessment?

23 A. Well, it was primarily done without

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

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

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

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

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

1 results from animal studies and,
2 primarily, these are based on results
3 testing whether PCBs cause cancer in
4 animals, and this is for any chemicals,
5 not just PCBs, and determine the
6 concentration in which that happens in
7 animals, apply a bunch of safety factors
8 to account for potential differences
9 between animals and humans, potential
10 differences within animals, etcetera, and
11 then come with a number which, in their
12 view, is protective of human health, so
13 they come up with this, in this
14 particular case it's called a cancer
15 slope factor. They apply that number to
16 a concentration to determine, in their
17 view, whether there is some theoretical
18 risk. The true risk, and EPA
19 acknowledges this, could be zero in the
20 risk in the way you and I think about
21 it.

22 Q. Right.

23 A. But their theoretical risk is what

1 determines these cleanup levels.

2 Q. Well, obviously, the issues here
3 are, in fact, PCBs carcinogens. Do they
4 cause cancer?

5 A. Yes.

6 Q. And I think there's a big
7 disagreement with that. I don't think
8 you and I would agree with that.

9 A. I believe we would disagree in
10 humans. I don't think we would disagree
11 in animals, but I think we would disagree
12 whether PCBs cause cancer in humans, yes.

13 Q. And I assume you are familiar with
14 the various clinical studies, if you
15 will, that have tried to relate PCBs with
16 cancer and other problems?

17 A. Yes, I am.

18 Q. What is it that can Solutia does
19 agree, if they would, that PCBs causes at
20 certain levels?

21 A. We believe that PCBs, at high levels
22 of exposure in humans, are associated
23 with dermal skin effects, possibly

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

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

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

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

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

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

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

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

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

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

20 Q. You're transferring them?

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

1 -- you know, that leaves the more highly
2 chlorinated ones in the vat, but it
3 doesn't change the chemistry of an
4 individual PCB molecule by simply adding
5 heat, and that's why they're good.

6 That's why they were used in many of the
7 applications they were used is because
8 they are stable under conditions of
9 heating.

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

16 A. Well, I mean, I know you don't want
17 a chemistry lesson or a PCB manufacturing
18 lesson, but all the PCBs were very
19 complex materials, very complex mixtures
20 of materials, and as you go through the
21 product sequence from 1221 to 1242 up to
22 1260 or 1268, then the number of
23 chlorines on the molecules in those

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

6 Q. Okay.

7 A. But now, there was no product that
8 was all what we call decachlorobiphenyl,
9 the highest biphenyl with ten chlorines
10 on a ring. We actually considered making
11 that and some people did make that as a
12 product, but that was a white solid as
13 opposed to many of the other materials.
14 That was a pure single compound, but that
15 material itself was a component of some
16 of the other mixtures and, most notably,
17 would have been a component of Aroclor
18 1268.

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

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

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

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

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

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

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

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

20 A. Primarily, yes.

21 Q. How large a vat?

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

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

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

6 A. Again, I don't know.

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

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

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

15 A. Not that I know of.

16 Q. Or drainage system?

17 A. Not that I'm aware of.

18 Q. Does Solutia --

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

1 Q. Kind of like burning something on
2 the stove and trying to scrape it out?

3 A. Yes, exactly. It's basically the
4 consistency of road tar, I mean very much
5 the same.

6 Q. Does Solutia admit or agree that
7 there have been PCBs that have escaped
8 from the Solutia facility?

9 A. Yes, I think we would agree that
10 there has obviously been, in drainage
11 pathways, some PCBs have moved from our
12 facility to off-site, yes.

13 Q. Is there any agreement as to the
14 quantitative amount?

15 A. I don't believe that's
16 determinable. I don't know who I would
17 be agreeing or disagreeing with. I've
18 never heard an amount, so, you know, I
19 don't know.

20 Q. Well, you probably, like me, have
21 seen documents that talk about several
22 pounds per day and that type thing?

23 A. Right.

1 Q. Is that something that we can agree
2 on or is that something that's still in
3 dispute?

4 A. Well, I think we can agree that
5 those documents exist and the numbers are
6 there. I think, knowing the state of
7 analytical chemistry at the time, there
8 is some uncertainty around those numbers,
9 but I think they certainly stand for the
10 proposition that PCBs were in those waste
11 waters.

12 Q. Okay. When you say there's a
13 certain -- the analytical chemistry was
14 not available at the time, what do you
15 mean?

16 A. Well, I'm just talking about the
17 uncertainties in the measurement whether,
18 you know, if you're getting -- measure
19 one hundred parts per million, is that
20 because that sample really has one
21 hundred parts per million or the sample
22 before it had one thousand parts per
23 million and it carried over into that

1 sample or a number of things.

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

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

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

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

23 A. Well, I believe we started -- I

1 think you probably have been through the
2 history, but the report of PCBs in the
3 environment really began back in late
4 1966 by some Swedish researchers and, you
5 know, there was a learning curve that
6 Monsanto and others had to get on and
7 climb to and it took us a couple of years
8 to get on that learning curve and develop
9 the analytical techniques, but I believe,
10 if I've got my numbers right, by '68, we
11 were actually taking samples in Anniston
12 and by '69, you know, waste samples and
13 water samples and by '69, we were
14 actually able to start making some of
15 those kinds of measurements that are
16 reported on the documents that you're
17 referring to.

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

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

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

10 Q. Right.

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

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

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

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

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

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

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

7 Q. Even prior to '93?

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

15 Q. You have two landfills; correct?

16 A. That is correct.

17 Q. And each landfill has a number of
18 cells?

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

1 Q. And you're not sure?

2 A. I mean, there's clearly a west
3 landfill and it had at least one cell,
4 but I don't know that there were a number
5 like there were in the south landfill.

6 Q. And when you say you basically seal
7 those, and that was in what, the
8 seventies?

9 A. Well, the PCB landfill was generally
10 closed in early 1972, 1973, but as we got
11 into what was called the RCRA, the
12 R-C-R-A process, the Resource Recovery
13 and Conservation Act then we had to take
14 additional steps on all of the landfills,
15 as did all chemical manufacturers, to be
16 sure that they were brought up to
17 compliance, so in the late 1980s,
18 additional work was done on closing those
19 landfills, so my confidence increases as
20 we move through those various stages.
21 But certainly, by the time of the RCRA
22 closure in '98, I believe there was,
23 other than just the little bit of soil

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

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

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

1 Q. When was it that you had to get the
2 storm water permits? When would that
3 have been?

4 A. I'm sorry, I don't know the answer.
5 I don't know when the MPDS process really
6 went in. I think it was the mid-1970s, I
7 think, but don't hold me to that.

8 Q. Okay. In reading your testimony,
9 Dr. Kaley, in the Abernathy case, you
10 made mention when asked the question
11 about human health studies that you did
12 not feel a study is necessary nor
13 appropriate based on the understanding of
14 the size of the population and you go on
15 to say that to do an appropriate
16 epidemiology study, it takes thousands of
17 people and you don't believe that there
18 are thousands of people exposed in the
19 Anniston community. Does that refresh
20 your memory about it?

21 A. I believe that's a fairly -- I mean,
22 I remember that question and I'm sure
23 that's what I said, if you have that

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

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

6 A. Yes.

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

12 A. Yes.

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

15 A. Yes.

16 Q. What blood levels have you seen?

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

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

1 in this testimony, a human health study?

2 A. No. I think the distributions are
3 generally -- I mean, you see, you know, a
4 large proportion, a majority of the
5 people are nondetects or certainly right
6 at the level of detection. You see
7 another group of people who are what
8 would be called normal background levels
9 throughout the United States, and then
10 there is a smaller group of people that
11 do have current or past exposure based on
12 the blood levels.

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

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

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

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

1 A. Right, what I would call a morbidity
2 study, right.

3 Q. And you're saying that a mortality
4 study is more appropriate than a
5 morbidity study?

6 A. Well, I think it's more appropriate
7 to make a final determination on
8 whether, for example, PCBs are associated
9 with human cancer or other major
10 diseases.

11 Q. What is the level that you would
12 consider quote "nondetect" unquote or
13 what is the level that is considered
14 nondetect?

15 A. Well, that depends on -- I don't
16 know. Without being flip, that depends
17 on the laboratory and what their
18 capabilities are. I mean, I think most
19 of the detections in the samples I have
20 seen from, you know, whatever litigation
21 have been, you know, three parts per
22 billion in blood and five parts per
23 billion in blood, but certainly, we know

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

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

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

18 Q. What is the normal background
19 levels?

20 A. Okay. That's a different question.

21 Q. Right. All right.

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

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

14 Q. All right.

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

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

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

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

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

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

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

3 A. Yes.

4 Q. They talk about mean serum level?

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

7 Q. Is that the average?

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

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

15 A. Up to twenty, yes.

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

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

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

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

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

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

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

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

6 Q. Right.

7 A. So my number, my three to six is,
8 what I'm saying, is the same as their
9 point nine to one point five. I don't
10 think there's a lot of difference in
11 their -- a factor or two is not a big
12 difference in my mind, and I'm looking at
13 a broader view. I'm not looking at the
14 two most recent studies. I'm saying over
15 the last decade or last fifteen years, if
16 you look at all those studies, so I don't
17 disagree with this, nor do I think it's
18 very different from what I'm saying, but
19 that's, again, that's the average.

20 That's the mean. That's my -- their one
21 point five is my three number. They are
22 still made up with people that could go
23 up to tens or twenties that help make up

1 that average.

2 Q. And there are people, then, you say,
3 that have twenty parts per billion in the
4 general population?

5 A. Certainly, to the data we have
6 available to us, that still seems to be a
7 reasonable number.

8 Q. And that might be because they were
9 more highly exposed than those who have
10 one point five?

11 A. And through their normal living
12 without any unique source of that
13 exposure, yes.

14 Q. Would you agree that a person who
15 has currently three parts per billion had
16 more, assuming he was living, had more
17 fifteen years ago than he has now?

18 A. No, I wouldn't. I absolutely
19 disagree with that.

20 Q. Why is that?

21 A. Why should they? I mean, if I've
22 got three now, I would say fifteen years
23 ago, my number was lower because, as I

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

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

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

16 Q. Is that true for people in Anniston?

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

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

1 number.

2 A. Okay.

3 Q. Do you agree that those people are
4 at a higher risk for a disease or problem
5 than one in Anniston that has only one
6 point five parts per billion?

7 A. The answer to that is no for a
8 number of reasons.

9 Q. Tell me those reasons.

10 A. Number one, I think the reason is
11 that, I mean, we already talked about a
12 potential disagreement, but certainly,
13 the view that I hold that PCBs are not
14 associated with long-term serious health
15 affects with the potential exceptions of
16 dermal effects and transient liver
17 effects. So I don't necessarily believe
18 -- I don't believe, at whatever level
19 your PCB level is, that you are at real
20 risk, not theoretical, not regulatory
21 risk, at real risk of developing disease
22 as a result of those blood levels, okay,
23 so that's number one.

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

7 Q. They are low background levels
8 within --

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

11 Q. The general population?

12 A. Yes.

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

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

18 Q. Okay.

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

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

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

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

23 Q. At all?

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

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

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

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

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

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

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

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

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

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

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

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

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

1 it's getting in, it's getting in.

2 Q. Right.

3 A. Whether it's inhalation or dermal or
4 ingestion, however it's getting in that
5 those are all ways that PCBs can get in
6 your body and there's no one that's
7 really different than any other, I don't
8 think.

9 Q. I take it you are familiar with the
10 various studies, Brown, Bertazzi, Sinks?

11 A. Yes.

12 Q. And I'm sure you have read those?

13 A. Yes, I have.

14 Q. And do you agree or not that those
15 studies do indicate an association of
16 exposure to PCBs and cancer, for
17 instance?

18 A. Okay. I believe that some of those
19 studies report associations between
20 purported or real exposure to PCBs and
21 some individual cancers on an individual
22 study basis, but if you look at all those
23 studies taken as a whole, you do not see

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

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

8 A. Or all cancers combined, yes.

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

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

15 Q. Gastrointestinal, and one was some
16 other cancer?

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

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

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

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

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

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

1 suggestions or there are associations we
2 just talked about.

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

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

17 A. Yeah.

18 Q. Okay.

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

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

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

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

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

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

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

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

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

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

23 Q. Well, for instance, CDC, would they

1 be more equipped?

2 A. Well, I guess I don't know what you
3 mean really by --

4 Q. I'm not sure I know what it means
5 either.

6 A. I know, and the risk assessments
7 that are being done, without getting into
8 a lot of detail, basically say here are
9 the potential exposures, given those
10 exposures and given these numbers that we
11 can calculate that relate to possible or
12 theoretic risk, I basically multiply my
13 concentration in the environment by this
14 potential risk number and come up with a
15 number that says this population has so
16 much risks and that risk is either
17 acceptable or it needs to be reduced by
18 doing something. You can do that for the
19 human and you can do that for
20 ecological. That's what risk assessment
21 is all about.

22 Now, if you're saying by human
23 risk assessment, I'm going to take this

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

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

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

23 They'll say it's additional

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

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

19 Q. What do you mean?

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

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

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

10 Q. Are you saying --

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

14 Q. There's other compounders that --

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

1 Q. Are you saying the risks that are
2 quote "associated with PCBs" is
3 theoretical?

4 A. In risk assessment, as applied it is
5 a risk -- it's a real risk to an animal.

6 Q. Okay.

7 A. Or it is a real end point? It's not
8 even a risk. You've got animals that are
9 sick or are dying and this isn't just
10 PCBs. You take that -- how much it
11 caused that animal to get sick and divide
12 it by ten and you divide it by ten again
13 and you divide it by ten again to come to
14 a factor where you're comfortable there's
15 enough -- you have put in enough
16 uncertainty factors in there to say okay,
17 way down here, a human exposed to this
18 level isn't going to get sick like this
19 animal up here at these very high levels
20 did. All this is uncertain in here.

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

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

10 Q. Meaning?

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

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

20 Q. So why do you do a risk assessment?

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

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

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

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

12 Q. Correct.

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

23 Do you come up with a number?

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

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

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

1 A. Right.

2 Q. What's that level?

3 A. It depends on the animal and it
4 depends on the end point you're talking
5 about. And I'm not going to be able to
6 give you numbers, but it takes, for
7 example, it takes a lot more to give a
8 rat cancer than it does to cause an otter
9 or a mink to have reproductive failure.

10 Q. Okay. And so you take that as being
11 known?

12 A. And there are all sorts of numbers.

13 Q. You take that to be known and you're
14 saying that you don't know the level, if
15 there is a level, that causes that same
16 problem to a human?

17 A. Yes, I'm saying that and I'm saying
18 that we have never seen those problems in
19 humans to know that level, but we will
20 all agree that, because we see it in
21 animals, we're going to apply all these
22 safety factors and we're going to clean
23 up to that level for humans. Or animals

1 may be involved.

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

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

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

23 Q. Okay.

1 A. I do not believe that the exposures
2 in a stone as evidenced by human body
3 burdens indicate that any of those
4 persons is going to develop a disease as
5 a result of that exposure.

6 Q. Okay. Now, I have the letter you
7 wrote back in April of 2000 to the ATSDR.
8 Do you remember that?

9 A. I do.

10 Q. Did you have any help in writing
11 this letter? Did you write this letter?

12 A. Yes, I wrote that letter.

13 Q. Okay.

14 A. A few people moved some comas.

15 Q. Sir?

16 A. A few people moved some comas, but
17 it is almost exclusively my work.

18 Q. Okay. And what did you review --
19 this may be such a broad question you
20 can't answer it. What all did you review
21 in order to write this letter and it was
22 the comments to the --

23 A. Believe me, I know what it was the

1 comments to.

2 Q. Health consultation.

3 A. Dated February 14th, 2000.

4 Q. Right.

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

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

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

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

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

1 to all of the comments that they
2 received.

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

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

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

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

16 Q. I may have.

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

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

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

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

10 A. That's correct.

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

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

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

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

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

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

5 Q. Okay.

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

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

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

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

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

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

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

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

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

1 they averaged to get those numbers out.

2 Q. Now, you also have some criticism
3 here about the extrapolations. "A
4 suggestion that back extrapolations can
5 be used to estimate historical PCB body
6 burdens in Anniston is over simplified."

7 A. I don't remember saying that, but
8 okay. I'm glad I did.

9 Q. "This discussion must be expanded to
10 elaborate on the uncertainties involved
11 in and introduced by this process,
12 speculative process," you call it, and
13 then you talk about "in the first place,
14 PCB half lives are congener specific."
15 What are the half lives of PCBs?

16 A. Well, let's go to what is a half
17 life.

18 Q. Okay.

19 A. All right, whether you're talking
20 about a chemical or a pharmaceutical,
21 whatever, if you put something in your
22 body and have some measure, then, of the
23 level of that thing in your body,

1 whatever it is.

2 Q. The dosage amount?

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

10 Q. Ten parts per billion?

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

1 that's a half life.

2 Q. Whether it be hours or days or
3 whatever?

4 A. Days or minutes, whatever. And then
5 if you start at that point, then one half
6 life later, you're only going to have a
7 quarter of it left so it's basically a
8 way of measuring how quickly things are
9 removed from your body.

10 Q. Okay. With that said, can you say
11 what the half life is of PCBs in
12 general?

13 A. I do not believe you can.

14 Q. Okay and that's because --

15 A. That's because there's no such thing
16 as a PCB. There are two hundred and nine
17 different things called PCBs that each
18 one is going to behave differently in a
19 human body and an animal body and they're
20 each one going to have an unique half
21 life.

22 Now, you can -- and people have
23 -- I'm not so naive as to say that

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

6 Q. Okay. And what is --

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

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

1 Q. So do you have any -- what is the
2 half lives of the general population?

3 A. I don't know.

4 Q. You have no way of knowing?

5 A. I know what has been measured in
6 capacitor workers.

7 Q. Which is?

8 A. I think for the lower, I'm guessing
9 it was one to two years and for the
10 higher, it was seven to eight years, but
11 that only applies to those capacitor
12 workers and it applies on a Hugh's
13 average and, you know, you can't do the
14 calculation. You can have the concept.
15 I don't have a problem with the concept
16 --

17 Q. Right.

18 A. -- as long as you can check for
19 background first.

20 Q. Right.

21 A. If you know someone who is exposed
22 and you know that that exposure blood
23 level is decreasing over time, then you

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

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

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

10 Q. Has that ever been studied or
11 measured?

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

23 Q. Okay. When you say doesn't really

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

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

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

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

14 Q. That's exactly what I --

15 A. Well, yeah.

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

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

1 it's meaningless anyway.

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

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

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

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

8 Q. And I think I have that.

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

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

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

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

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

13 Q. That it got there some other way?

14 A. Correct.

15 Q. Fill dirt?

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

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

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

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

5 Q. Okay.

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

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

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

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

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

23 Q. What way would the property outside

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

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

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

1 guess, from, you know, hearsay and
2 speculation, but that would be my answer
3 to your question.

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

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

12 Q. I'm looking for the map that
13 describes the areas and I think you said
14 one, two, three, four, five, six, and I
15 --

16 A. I can use a break --

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

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

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

9 (Whereupon, a short recess was

10 taken at this time.)

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

15 A. Yes.

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

19 A. No.

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

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

23 Q. What term would you rather use?

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

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

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

11 Q. What are coplanars?

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

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

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

7 Q. Do they still rotate?

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

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

1 reception, it can bind to that receptor.

2 That's why it matters whether they are

3 coplanar or not.

4 Q. That's why some people call it

5 dioxin-like?

6 A. Yes, because the theory is, and it

7 is a theory, is that once a PCB congener

8 can bind into that receptor, it could

9 then trigger those same responses that

10 dioxin would, although at much lower

11 levels or much lower potency.

12 Q. Okay. And why is it that you do not

13 agree that that can occur; that is, that

14 it can bind to the AH receptor?

15 A. I totally agree that the binding can

16 occur. I do not disagree that the

17 binding can occur. What has never been

18 shown in the laboratory is all the rest

19 of that cascade of physiological or

20 toxicological events that have been shown

21 for dioxin. No one has ever really shown

22 that few, if any, of those have ever

23 occurred by the binding of a PCB molecule

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

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

5 A. Yes.

6 Q. Which ones are those?

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

16 Q. Okay.

17 A. Those are four totally coplanar
18 PCBs.

19 Q. All right.

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

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

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

21 A. I think that has never been shown.

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

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

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

19 Q. That's a known?

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

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

5 Q. Is that going to come out before
6 October?

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

9 Q. Okay, just asking.

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

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

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

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

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

22 A. No, no, no. Rats.

23 Q. There are animal studies that --

1 A. That show that TCDD, a single --
2 not like PCBs, there are, you know,
3 seventy-five dioxins.

4 Q. There's one dioxin and it causes
5 cancer in rats?

6 A. Yes.

7 Q. What's the difference between that
8 study and the study that shows PCBs cause
9 tumors or cancers in rats?

10 A. Well, I'm not sure what your
11 question means. I mean, it's a different
12 chemical. I suppose it's as DDT causes
13 cancer in rats or something else. The
14 problem really is that dioxin does it at
15 so much lower levels.

16 Q. Okay. That's what I'm saying. We
17 know that there are animal studies that
18 show an association with --

19 A. I think in animal studies we've got
20 all that controlled. I'll go with show.

21 Q. PCBs show that there's cancers in
22 rats?

23 A. Yes.

1 Q. We also have studies that show
2 dioxins cause --

3 A. A dioxin causes cancer.

4 Q. What my question is, I guess, is,
5 all right, how can you take the giant
6 leap from the dioxins in rats causing
7 cancer in humans and you can't take a
8 giant leap with respect to PCBs causing
9 cancer in rats to humans? Why can't you
10 take the same leap?

11 A. I don't take the first leap. I
12 would never take the first leap. You
13 can't take either leap.

14 Q. So you won't agree that, based on
15 the animal studies of dioxin causing
16 cancer in rats that that proves or shows
17 that dioxin causes cancer in humans?

18 A. Oh heavens, no. I mean, we would
19 have exactly the same discussion around
20 the dioxin-human lecture that we do with
21 the PCB. What I was trying to say is
22 that TCDD causes cancer in rats.

23 Q. Okay.

1 A. PCBs -- A PCB mixture, Aroclor 1260
2 causes cancer in rats, but the in-between
3 is one teeny component of that Aroclor
4 1260 has never been tested to determine
5 whether it causes cancer in rats, so we
6 can't make the leap that, you know,
7 because that little, you know, we don't
8 know the mechanism of really how dioxin
9 causes cancer in rats or how PCBs cause
10 cancer in rats.

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

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

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

3 A. Okay, and I don't.

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

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

17 Q. Ramifications?

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

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

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

3 A. Yes.

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

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

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

20 Is there the same problem with those
21 studies?

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

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

9 Q. Okay.

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

22 And there are huge problems
23 with confounding in those studies because

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

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

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

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

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

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

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

1 mixture back to dioxin.

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

6 A. Correct, the ones we talked about
7 earlier.

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

10 A. Right, truly coplanar, right.

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

13 A. The little swiquets.

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

16 A. That's correct.

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

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

1 help them economically assess risks for
2 cleanup sites.

3 If you have a complex mixture
4 of dioxins or other, you know,
5 dioxin-like materials, arguably, to do a
6 risk assessment on that site, you would
7 have to go in and measure every compound,
8 have toxicity studies on every compound,
9 and assess independently the risks of
10 every compound in that mixture, and then
11 somehow add all those up. So everybody

12 -- and I would agree that's -- we don't
13 have the toxicological information. It
14 would be years away and very costly to
15 get the toxicological information.

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

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

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

12 Q. Why?

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

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

21 A. Right, even in animals.

22 Q. Even in animals?

23 A. Even in animals to do that.

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

8 Q. Total?

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

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

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

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

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

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

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

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

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

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

5 A. I don't think I said that.

6 Q. Maybe I better read it correctly.

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

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

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

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

22 A. Right.

23 Q. But you're saying that there were no

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

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

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

11 A. Okay, now --

12 Q. Are we talking two different things
13 here?

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

16 Q. Okay.

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

1 dibenzofurans or on short, furans.

2 Q. Right.

3 A. All right, there's another group of
4 chemicals called PCBs.

5 Q. Okay.

6 A. So we've got these three big groups
7 that we're all going to be talking about.
8 Now, within the PCB group, there's this
9 little teeny twelve-member little
10 subgroup called dioxin-like as you refer
11 to it and others are the coplanars. So
12 your question was dioxins or coplanars
13 and they're separate answers to those two
14 questions.

15 Q. All right, that's maybe my confusion
16 in reading this letter when you say PCDDs
17 or dioxins are not produced as
18 by-products for the manufacture of PCBs,
19 nor are they formed by thermal stress of
20 PCBs.

21 A. Right. That's that one group of
22 specific compounds, right.

23 Q. "Therefore, the PCDD or dioxin levels

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

4 A. Right.

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

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

16 Q. Okay.

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

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

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

7 A. That's correct, I was not.

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

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

14 Q. Well, the reason I'm asking that is,
15 in the same paragraph, you're mentioning
16 the TEQs and the dioxin and furans, and
17 that's what's confusing me some because I
18 --

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

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

4 Q. All right.

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

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

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

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

5 A. Yes.

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

10 A. Yes.

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

17 A. Right.

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

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

22 Q. Right.

23 A. The second number is total PCBs.

1 Q. Right.

2 A. Third and fourth column are the sum
3 TEQs for dioxins, furans, and PCBs in
4 blood where the third column is without
5 the PCBs included and the fourth column
6 is with the PCBs included.

7 And when I'm talking
8 specifically here about that, you know,
9 maybe I wasn't clear here, but I was
10 talking primarily about the withouts.
11 I'm talking about the TEQs from the
12 dioxins and furans, not from the PCBs.

13 Q. All right.

14 A. Now, that person, because he has
15 higher PCBs, I think it's number one,
16 obviously, if it's the highest, is going
17 to have, with the PCBs, a higher number
18 than the others also because he's got
19 more PCBs, so that would just make sense.
20 But that comment was really addressed to
21 this without the PCBs column --

22 Q. Okay. And then you mention the --

23 A. -- because I think I say one

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

4 Q. I think you did somewhere.

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

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

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

16 Q. What was her level of PCBs?

17 A. Of PCBs?

18 Q. Right, number nine?

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

21 Q. And her TEQs?

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

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

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

8 A. Because her OCDD level is very high.

9 Q. Okay.

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

16 Q. How would you explain that?

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

1 analytical error. It's just sticks out

2 so badly. I can't -- you know, in my

3 comments, I couldn't speculate on it.

4 I'm just saying it sticks out.

5 Q. All right. On page fourteen of your

6 letter, I think you're talking here about

7 some, I guess, soil samples, yes.

8 "Solutia suggests that the agency avail

9 itself of all avenues to ascertain the

10 conditions under which samples were

11 collected by agents of the plaintiffs'

12 attorneys. It is Solutia's understanding

13 that many of the samples were collected

14 in areas calculated to maximize potential

15 soil levels such as downspout." Can you

16 tell me who you're referring to?

17 A. To what plaintiff's attorney I'm

18 referring to?

19 Q. No, agent.

20 A. I mean, those data were all supplied

21 by the plaintiffs' attorney in the

22 Abernathy litigation, so I'm talking

23 about whoever collected the samples.

1 Q. You don't know particularly who that
2 was, not attorney but --

3 A. I'm tempted to say Mr. Bonner or Dr.
4 Bonner, but I don't know that for a
5 fact. I could be wrong on that. I'm
6 tempted to say that's who it was and,
7 again, let me -- I'm not saying he did
8 anything wrong.

9 Q. I understand.

10 A. I'm just saying it wasn't an average
11 value. My understanding was that they
12 went to areas on the property where they
13 expected the levels to be the highest.

14 Q. Well, you go on to say that "our
15 understanding is based on the deposition
16 testimony of one of the persons who
17 performed soil sampling for the larger of
18 the two plaintiffs' groups. For
19 instance, we will provide relevant
20 sections of that deposition to ATSDR upon
21 request."

22 A. Yes, and I don't think that was ever
23 requested and I don't think we did.

1 Q. You don't know who you're referring
2 to?

3 A. I think it was Dr. Bonner.

4 Q. Dr. Bonner?

5 A. I think so, but I could be wrong.

6 Q. Okay. Now, any area you refer in
7 here as under other pathways, you say
8 that in Monsanto, in 1970 or '71,
9 Monsanto became aware that hogs were
10 being raised on or near Monsanto property
11 on an area that had been used for waste
12 disposal. Can you specify what area you
13 -- do you know what area you're talking
14 about?

15 A. It was somewhere on the south
16 landfill. More specifically than that, I
17 don't know. That's my understanding.

18 Q. The hogs rooting around in this
19 secluded area were possibly being exposed
20 to waste materials. Do you have any
21 information as to whether or not these
22 hogs were analyzed as far as PCB levels?

23 A. I believe they were not.

1 Q. Was there ever any time that logs
2 were analyzed?

3 A. My understanding is I think it's
4 covered by people's testimony. My
5 understanding is that a hog was found
6 dead on Solutia property, that its PCB
7 levels were measured and found to be
8 elevated, and that, based on that, people
9 from the plant found that there were
10 other hogs on the property and that those
11 hogs were purchased but were not tested.

12 Q. Was there any analysis done on the
13 hog that was, in fact, the initial hog on
14 the liver?

15 A. I don't know what was -- I do not
16 know what was specifically tested. I
17 don't know. I think the fat was, but I
18 don't know whether the liver was or not.

19 Q. And I think y'all found the map or a
20 map?

21 A. Yes.

22 MR. KELLY: Can we go off the
23 record?

1 MR. RODEN: Yes, okay.

2 (Whereupon, a short discussion
3 was held off the record at
4 this time.)

5 MR. RODEN: Make a note we're
6 going to mark Exhibit No.
7 1 and we're going to
8 substitute it for a copy.

9 (Whereupon, Plaintiff's Exhibit
10 No. 1 was marked for
11 identification.)

12 Q. All right, Dr. Kaley, just for
13 purposes of identifying, just tell me
14 what we are looking at? What is this?

15 A. This is a map supplied by the
16 Environment Protection Agency which
17 designates by number with one exception,
18 one of them is by letter, areas in the
19 north and east of the Monsanto plant
20 which were to be tested for -- which
21 residential properties were to be tested
22 to determine if PCBs were present in
23 those properties in five point composite

1 samples above ten parts per million.

2 Q. And is this the same legend that has
3 been attached to the proposed order as
4 pending?

5 A. This is an attached -- this is part
6 of an attachment to the proposed consent
7 decree, but it is -- what's really
8 attached to the proposed consent decree
9 is the administrative order of consent
10 dated -- I think it was October 3rd, 2001
11 to which this is an attachment or an
12 exhibit, so it's an attachment to an
13 attachment.

14 Q. Okay. What was the purpose, again,
15 of this particular map?

16 A. The purpose of this map was to
17 delineate and prioritize areas for
18 sampling for Solutia under the terms of
19 the consent order.

20 Q. And then the proposal, its function
21 is the same under the proposed consent
22 decree?

23 A. I don't think so. I think -- well,

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

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

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

4 Q. And you sampled how many?

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

20 Q. Okay.

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

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

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

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

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

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

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

23 Q. And it would be true on the consent

1 decree, it would follow a map whether

2 it's this map or another map?

3 A. I believe that would be correct.

4 Q. So if a piece of property is outside

5 these zones, would they ever be tested

6 under the consent decree?

7 A. You're stretching my knowledge of

8 the consent decree. My belief is that

9 they would not be.

10 Q. Okay.

11 A. Now, can I make just one more

12 addition just to clear things up?

13 Q. Sure.

14 A. There is one additional area that is

15 not shown on this map. It is called Area

16 OLN, all caps, which stands for Oxford

17 Lake Neighborhood and that's a little

18 area down by the Oxford Lake softball

19 complex down off Highway 78 so that's

20 also included in the consent order, but

21 it's not on this map.

22 Q. What prompted that?

23 A. The discovery of a couple of yards

1 there that had PCBs in them.

2 Q. Was that through some litigation?

3 A. No, it's part of our investigation

4 along our -- well, I think it was

5 actually prompted by a request by one of

6 the residents there to have his property

7 sampled because we had been doing work

8 along that neighborhood and my

9 recollection is he had taken sediments

10 from the creek and put them up in his

11 yards, so we tested an additional group

12 of properties down in that area.

13 Q. What is the significance of the zone

14 "F"?

15 A. I don't recall right offhand. I

16 think that's an area near Quintard Mall,

17 but, again, I could be wrong, and why

18 "F", I have no clue.

19 Q. This map is entitled "Zones and

20 Drainage Area for Remediation".

21 A. Yes.

22 Q. And again, I have asked you this

23 once before; do you have any knowledge of

1 the basis for these zones?

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

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

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

6 Q. That's what I assumed, but I wanted
7 --

8 A. It's clearly drainage pathway
9 driven.

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

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

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

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

1 really can't generalize.

2 Q. Under the consent decree, the
3 proposed consent decree, would all
4 properties in these zones be tested?

5 A. Probably not each property. Going
6 back to what we discussed earlier, that
7 as we gather more and more information by
8 our sampling, it is possible,
9 statistically, to make some predictions
10 about whether PCBs are likely or not
11 likely to be in a given area and, subject
12 to EPA's approval, we can terminate
13 sampling in a given zone if we can
14 statistically demonstrate to the EPA's
15 satisfaction that we're not likely to
16 find PCBs in those yards.

17 Q. But as you start the process, let's
18 say you start in Zone 1 --

19 A. I'm not sure which one was first.
20 Two and three were clearly the highest
21 priority.

22 Q. So not No. 1?

23 A. No. These are not labeled by

1 priority.

2 Q. So two and three is considered high
3 priority?

4 A. I believe. I think that's what it
5 was. It says it in the consent decree
6 specifically.

7 Q. Would you necessarily test every
8 piece of property in zone two and three?

9 A. No, you would test every piece of
10 property nearest the drainage things,
11 moving away, and if you got to the point
12 to where we and the agencies were
13 comfortable that we weren't finding PCBs
14 anymore, we could terminate even in those
15 high priority zones.

16 Now, I believe, and, again, I'm
17 moving a little further than I should, I
18 think we've only terminated samples in
19 one zone and I don't remember which one
20 it is. Otherwise, we have not been able
21 to demonstrate that is my recollection.

22 Q. You don't know if it's No. 1 or --

23 A. I believe it's No. 3, but I don't

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

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

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

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

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

10 Q. Again, that may be Mr. Branchfield?

11 A. Clearly. Clearly, Mr. Branchfield.

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

15 A. Yes.

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

20 A. That would be by my understanding, yes.

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

1 A. Million.

2 Q. Million, excuse me.

3 A. With an "M".

4 Q. -- with an "M", be cleaned up?

5 A. We would make the offer to clean it
6 up.

7 Q. And what would be the procedure for
8 cleaning it up?

9 A. Removal of, basically, the top one
10 foot of soil from the property and
11 testing to be sure that some level isn't
12 present underneath that one foot and then
13 the one foot would be replaced with clean
14 soil and revegetated.

15 Q. So if you clean the one foot off and
16 then you test it again and still have one
17 part --

18 A. No, I think it's ten at the one-foot
19 level. I think it's ten.

20 Q. Okay. It goes to ten?

21 A. It goes to something else. I think
22 it's a ten and that's consistent with
23 EPA's spill cleanup policy for

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

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

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

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

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

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

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

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

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

1 superimposed on these zones or not.

2 Q. Okay. The Tox Profile we talked
3 about earlier, I'm sure you're familiar
4 with?

5 A. Yes.

6 Q. And I assume you know -- is it Dr.
7 Copland?

8 A. I mean I know who he is. I've seen
9 him. I've heard him speak. He certainly
10 wouldn't know me. Oh, he's written me a
11 letter so he may know me.

12 Q. Tell me, what is, in fact, the Tox
13 Profile? What is it for?

14 A. Under the terms of the act creating
15 ATSDR, the Agency for Tox Substance and
16 Disease Registry, one of the things which
17 is actually funded by EPA's superfund
18 money, one of the things they were tasked
19 to do was prioritize chemicals that were
20 found at superfund sites.

21 Q. Okay.

22 A. So they have got lists of how many
23 superfund sites benzene was found or

1 asbestos was found or PCBs were found,

2 okay. That lists exists.

3 Q. I think I have that somewhere.

4 A. Yes, and it's almost universally

5 misinterpreted. Okay, based on that

6 list, EPA or ATSDR is then commissioned

7 to either do or hire contractors to do,

8 which is what they always do, an

9 expensive literature review of everything

10 that is known or everything that is found

11 in the literature about, primarily, the

12 toxicity of that particular compound, so

13 there are dozens if not hundreds of

14 toxicological profiles for a wide variety

15 of chemicals, so that's what they are.

16 This is one in a huge series.

17 Q. And --

18 A. And they are periodically revised.

19 I think this is the third revision of the

20 PCB Tox Profile.

21 Q. Is this the current --

22 A. It's the most recent, yes.

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

1 this earlier, it says it's peer
2 reviewed.

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

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

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

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

1 Q. The reason I asked, is this an
2 authoritative document for people such as
3 you to look at to rely on as an
4 authoritative?

5 A. To the extent that the document is a
6 compilation of literature information on
7 toxicity, environmental behavior,
8 environment levels, a number of things
9 out of the literature that were reviewed
10 incorporated, yes, I would agree that it
11 is an authoritative document and is
12 relied upon and I rely upon it to the
13 extent that interpretations based on that
14 literature are superimposed on the
15 factual basis of the document. I have
16 some problems with the way things, some
17 of the things that are said, but usually,
18 more the way they are said.

19 Q. Okay, well let me just ask you, it
20 its says here under Summary of Health
21 Effects, Chapter Two, "Health effects
22 that have been associated with PCBs in
23 humans and/or animals include liver,

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

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

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

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

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

23 (Whereupon, a short recess was

1 taken at this time.)

2 Q. I think I've gone over this, Dr.

3 Kaley. Let me ask you one thing. This

4 is the letter or, excuse me, the health

5 consult of February of 2000 and I think

6 we've talked about this, but on page six

7 my copy which is, you're right, it's off

8 the internet, so it's going to be

9 different, but when they talk about the

10 persistency of PCBs and the half lives of

11 PCBs and they talk about the fact that if

12 one -- let's see, talking about the range

13 of half lives being from what?

14 A. Three to twenty four.

15 Q. Three to twenty-four, and basically

16 say that if one has a level today, their

17 level fifteen years ago would be twice as

18 high or something like that. You don't

19 agree with that statement?

20 A. No.

21 Q. I think you voiced that in your

22 letter; correct?

23 A. Well, I think the most important

1 thing in this paragraph is the chain of
2 ifs.

3 Q. Okay.

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

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

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

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

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

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

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

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

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

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

17 Q. Meaning, again, nondetect meaning
18 what?

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

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

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

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

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

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

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

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

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

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

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

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

3 A. Yes.

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

5 A. Yes.

6 Q. Is that the main source of that?

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

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

15 A. Right.

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

20 A. Right.

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

23 A. Yes.

1 Q. That's pretty high, is it not?

2 A. For a residential property, yes, I
3 would admit that's elevated.

4 Q. And in the range of the top ten, I'm
5 not sure I understand the range,
6 seventeen point four to eight hundred
7 forty?

8 A. Well, if you rank them one through
9 six hundred fifty-five, number one was
10 eight hundred forty and number ten was
11 seventeen point four and they went down
12 from there.

13 Q. Okay.

14 A. That's the range of the top ten
15 samples, so obviously, it drops real
16 fast.

17 Q. Okay.

18 A. But I don't whether that eight
19 hundred forty or the seventeen was in or
20 out of the flood plain. That's why I
21 can't answer your question. I don't know
22 where those samples were. Those top ten
23 may have very well been all in the flood

1 plain.

2 Q. You don't remember that?

3 A. I don't know if -- no, I doubt if I
4 did know at one time.

5 Q. Now, I recall seeing something and,
6 again, it may have been a newspaper
7 article, I can't tell you, but, something
8 about Solutia and/or Monsanto using
9 mercury in their processing?

10 A. Yes.

11 Q. What exactly would that use mercury
12 for?

13 A. Mercury was used in the process --
14 we made chlorine at the Anniston plant
15 from the mid-1950s, I want to say 1955 to
16 1969 and the process we used is an
17 electrical chemical process and mercury
18 was used as one of the electrodes in that
19 process.

20 Q. Did you say '55?

21 A. Yes.

22 Q. To '69?

23 A. I believe it's that general time

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

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

6 A. Yes. That was certainly the intent.

7 Q. Meaning it was recirculated or
8 recycled or whatever?

9 A. Yes.

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

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

18 Q. And when were those efforts made?

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

20 Q. And was that successful?

21 A. To a large extent, yes.

22 Q. What do you mean?

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

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

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

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

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

5 Q. What was their estimates; do you
6 recall?

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

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

11 A. Yes.

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

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

1 elevated levels.

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

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

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

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

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

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

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

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

1 Q. What about the loss or was there any
2 lead used in the process?

3 A. For a short period of time, there
4 was lead used in the process to make
5 biphenyl which is the starting material
6 for PCBs. It was basically done in lead
7 pots.

8 Q. And in what period of time?

9 A. I should know, but I don't
10 remember. Probably started -- it was
11 probably from early on and I think we
12 started changing those out in the
13 mid-fifties, but as I'm sitting here
14 today, I don't remember the dates on
15 that.

16 Q. Do you remember whether or not there
17 was an effort made to prevent the escape
18 of the lead into the environment?

19 A. Yes. Well, part of it -- I mean it
20 was called a lead pot process and there
21 was really very little chance for lead to
22 escape in the environment anyway, but,
23 again, my recollection of my review was

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

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

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

8 Q. Has that been questioned in the
9 recent past?

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

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

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

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

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

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

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

1 about that profile.

2 "Carcinogenesis of PCBs in
3 humans has been investigated in
4 retrospective occupational studies that
5 evaluating cancer mortality in worker's
6 exposure in capacity, manufacturing, and
7 repairing," and I think that refers to
8 the Bertazzi and the Brown and the other
9 studies, I assume?

10 A. Yes.

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

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

23 A. I would agree with that in the terms

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

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

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

20 The first criteria is animal
21 carcinogenicity and if something is an
22 animal carcinogen, it automatically
23 becomes a probable human carcinogen in

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

3 Q. Okay.

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

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

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

1 is that ninety nine percent?

2 A. No. I don't know what it is. In
3 IRAC's assessment, in general, it means
4 that -- they just basically put together
5 a group of scientists and that group of
6 scientists, some of them look at the
7 animal data and some of them look at the
8 human data and if the group looking at
9 the human data does a weight of the
10 evidence assessment concludes in their
11 minds that there's enough human data on
12 that compound, there are enough positive
13 studies, there's consistency, there's
14 strength of association, all those
15 Bradford Hill criteria, I'm sure you have
16 heard about until your ears are full,
17 then they will make that determination.

18 Q. That it is --

19 A. That it is a known human -- that the
20 human information is conclusive. I think
21 the term they actually use is "adequate",
22 and that it is a known human carcinogen.

23 Now, we have talked about

1 dioxin, and they bent their rules on
2 dioxin because the human epidemiology
3 group decided that there was not enough
4 information on dioxin to call it a known
5 human carcinogen, but the overall
6 committee -- I'll save my rhetorical or
7 editorial comments -- the overall
8 committee decided that there was enough
9 known about the mechanism of the cancer
10 in animals and the fact that that
11 mechanism probably occurred in humans
12 that they were going to name it a known
13 human carcinogen even in the face of
14 inadequate information, so they kind of
15 tweaked the --

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

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

1 Q. And if it's suggestive, it becomes
2 probable?

3 A. It remains probable.

4 Q. Okay. So you may have animal
5 studies that show --

6 A. Are conclusive.

7 Q. Are conclusive, so therefore, it's a
8 probable causation?

9 A. Yes, automatically.

10 Q. If you have human data, it becomes
11 suggestive?

12 A. It can be. I mean, you can have
13 inadequate data which says there's really
14 nothing there or there could be negative
15 human data which would probably still
16 leave it a probable or you could have
17 what they call the suggestive which is,
18 again, in the interpretation, I'm not
19 sure that I would agree with their
20 interpretation even under suggestive.

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

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

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

7 A. No.

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

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

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

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

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

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

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

19 Q. And by whom?

20 A. By IARC and the US EPA.

21 Q. And IARC is who?

22 A. International Agency for Research on
23 Cancer.

1 Q. And who exactly is that?

2 A. It is what it is. I think it's

3 associated with the World Health

4 Organization, WHO, and it's basically a

5 group that that's what they do is they

6 get together and talk about the

7 information available on candem. The

8 primary purpose is to say okay, we've got

9 "X" resources we can spend on cancer

10 research, how are they best spent, well,

11 let's not waste them on things that are

12 non-carcinogens and maybe let's not waste

13 them on things that are known carcinogens

14 because we already know that, let's think

15 about what research could we do on

16 something that we need to learn more

17 about. That's really the kind of purpose

18 of their classification is to guide

19 research into those areas.

20 Q. Does that same agency classify

21 dioxins as known carcinogens?

22 A. IARC classifies dioxins as known

23 carcinogens. I just talked about the

1 process by which they did that.

2 Q. And as does EPA?

3 A. EPA is trying to do that. They have
4 not. That is one of the controversial
5 proposals of their still draft dioxin
6 reassessment.

7 Q. And as I understand from this
8 afternoon's discussion with you, you
9 don't agree that dioxins even are a known
10 carcinogen?

11 A. I don't agree that the human
12 evidence for dioxin carcinogenicity in
13 humans is sufficient to classify it as
14 known.

15 Q. So you don't agree with IARC;
16 correct?

17 A. I don't agree that dioxin causes
18 cancer in people.

19 Q. And you don't agree with IARC as it
20 relates to PCB being a probable
21 carcinogen or do you?

22 A. I agree that it meets their
23 classification for probable carcinogen. I

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

8 Q. And it's only positive in animals?

9 A. Pardon?

10 Q. And it's only positive in animals?

11 A. It's positive in only animals.

12 Q. That's the distinction?

13 A. Yes.

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

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

23 Q. Okay.

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

4 For this deposition
5 specifically, I reviewed my -- I have an
6 affidavit, kind of a historical summary
7 affidavit that has been filed in a number
8 of cases. I did review that.

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

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

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

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

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

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

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

4 A. It depends on which ten.

5 Q. Correct.

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

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

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

1 a factor to be a difference, so I think
2 there's some reconciliation that needs to
3 be thought about in some of those data.

4 I don't have any basic objections that
5 one is right and one is wrong, here's why
6 it's right and here's why it's wrong.

7 Q. Okay. You just observe some
8 discrepancies --

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

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

23 One of the things I don't know

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

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

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

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

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

21 A. Probably.

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

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

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

9 A. Yes.

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

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

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

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

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

1 can recall?

2 A. That's a very broad question. I
3 believe that some of our experts are
4 physicians and with some of our experts,
5 I have probably had some discussion about
6 the health of this plaintiff group or
7 others, but I don't have any specific
8 recollections of that. I mean, is that
9 what you meant?

10 Q. Well, as the director of
11 environmental affairs for Solutia, is it
12 part of your job to analyze or determine,
13 if you will, the health effects or
14 possible health effects on the community
15 from PCBs?

16 A. I would say that's part of my
17 responsibility, yes.

18 Q. And as part of that responsibility
19 to do that, do you, on a regular or
20 frequent or other basis, consult with
21 physicians to keep abreast of what, if
22 any, health effects there might be
23 associated with exposure to PCBs?

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

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

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

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

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

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

4 Q. Well, the literature first?

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

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

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

23 I'm sure you get tired of

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

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

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

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

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

23 Q. And if that were to happen, if you

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

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

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

14 A. I have no idea.

15 Q. Has that ever been discussed with
16 Pharmacia?

17 A. Not by me or with me present.

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

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

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

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

9 A. Not that I'm aware of.

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

13 A. I'm sorry?

14 Q. Has anyone left Solutia --

15 A. Has anyone left Solutia to go to
16 Pharmacia?

17 Q. Yes.

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

20 Q. I think you mentioned the dioxin
21 reassessment.

22 A. Yes.

23 Q. What exactly is that?

1 A. A very long story. I'll be brief.

2 It is an attempt by the EPA to do a

3 comprehensive reassessment of all the

4 toxicological information and human

5 health information known about,

6 primarily, TCDD, but encompassing the

7 broader category of what we've jokingly

8 been calling dioxin-like compounds in and

9 effort to provide information for risk

10 assessors and risk managers about what to

11 do with dioxin contaminated cleanup

12 sites, primarily, but also having

13 implications for dioxin levels in the

14 human population.

15 Q. And it addresses the areas of the

16 dioxin-like PCBs?

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

18 does.

19 Q. And does it conclude that the

20 coplanars or dioxin-like PCBs are similar

21 to the dioxins or the TCDD that causes

22 cancer?

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

1 think you were going --

2 Q. Okay, thank you.

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

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

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

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

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

7 Q. Why?

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

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

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

18 Q. They got outvoted?

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

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

3 Q. And who are the members of that --

4 A. That has not been established. The
5 committee has not been established.

6 There is merely a proposal that it be
7 reviewed by the National Academy of
8 Sciences.

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

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

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

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

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

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

10 A. No, I do not believe there is.

11 Q. Are you familiar with Maroni?

12 A. Yes.

13 Q. And is it Fishbein?

14 A. Fishbein.

15 Q. Do those studies not indicate --

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

21 Q. Not robust enough is your --

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

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

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

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

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

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

19 Q. Okay.

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

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

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

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

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

15 A. No, I haven't.

16 Q. Have you ever had a liver profile
17 done?

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

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

22 A. Not that I know of.

23 Q. Do you know of anyone that works for

1 Solutia that has had their serum level
2 for PCBs performed?

3 A. Yes.

4 Q. Okay. Who?

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

6 Q. Is that something that has been
7 routinely done?

8 A. No.

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

12 A. No.

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

22 A. Monsanto workers or workers --

23 Q. Monsanto workers?

1 A. Oh, I don't know. I'm not aware of
2 anything.

3 Q. I think my question seemed to
4 indicate I was talking about the blood
5 serum, but what I meant to say was their
6 liver enzymes.

7 A. I'm not aware of that either.

8 Q. Okay. Since the Abernathy trial or
9 since you testified in the Abernathy
10 trial -- it's still going -- have you
11 given your testimony in any other case,
12 PCB-related case?

13 A. No.

14 Q. And I believe you testified in March
15 about year ago; is that your
16 recollection?

17 A. That's the latest time, yes. I
18 mean, I testified in the Gadsden phase
19 and the environmental -- what's the word
20 I'm looking for -- injunctive relief
21 phase, so I know two instances of my
22 testimony in that litigation. I believe
23 this is my first deposition in a case

1 since that time.

2 Q. Are there still ongoing
3 communications with the EPA and
4 yourself? Is there something going on
5 that y'all routinely communicate?

6 A. Not me. Now, Craig does. I mean --
7 well, let me say we are continuing to
8 submit documents in anticipation of the
9 eventual entering of the consent decree
10 and I review those documents, but there
11 is nothing going to the EPA under my
12 name.

13 Q. What about the Alabama Department of
14 Environmental Management, have you had
15 constant contact with them?

16 A. Not recently, no.

17 Q. There's a letter here dated -- I
18 don't know where I got it.

19 A. Wait, stop, I do, because we have to
20 report on a bimonthly basis air levels
21 from our air monitoring program to ADEM
22 and EPA and I write those letters, so
23 yes.

1 Q. March 10th of 2003; is that the most
2 recent letter?

3 A. That's the most recent.

4 Q. Okay. What's the purpose of your
5 having to write them?

6 A. Do you want a twelve-year-old kid's
7 answer?

8 Q. A short answer.

9 A. They told us to. No, we have been
10 doing air monitoring. We had proposed to
11 stop air monitoring because we don't
12 believe it is providing a whole lot
13 useful information. They have requested
14 that we continue to do that air
15 monitoring and provide them with the
16 results.

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

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

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

7 A. Me, personally?

8 Q. Right.

9 A. That's correct.

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

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

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

1 ordered, it would be Branchfield?

2 A. Branchfield, yes.

3 Q. And did you have any, I guess, input
4 into the actual drafting of the proposed
5 order itself?

6 A. Some.

7 Q. But would he have more input than
8 you?

9 A. Yes, but, by far, the EPA had a lot
10 more input than any of us.

11 Q. And what do you see as your, other
12 than a consulting role, what do you see
13 as your role in implementing the proposed
14 consent decree?

15 A. Other than advisory consulting,
16 nothing. I mean, you know, I'll continue
17 to review documents that we submit. I'll
18 continue to review things coming the
19 other direction, but other than that,
20 nothing.

21 Q. Who will actually do the -- under
22 the proposed consent decree, who will
23 actually do the actual soil sampling and

1 testing?

2 A. Our contractor to Solutia. We hire
3 people to do those things.

4 Q. Is there a particular one that you
5 use?

6 A. There's one we have been using.

7 Q. Is that one you think you will
8 continue using?

9 A. Probably.

10 Q. Who is that?

11 A. It's Genesis Project, Mike Price. I
12 need to take two seconds or two minutes.

13 Q. Okay. Let's go off the record a
14 minute.

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

17 Q. With respect to the cleanup or
18 assuming the soil samples indicate
19 there's one part per million, who
20 actually does the reclamation or
21 remediation?

22 A. We've got us a contractor. I don't
23 know -- I don't really know who's doing

1 that.

2 Q. So is that a bid-out job type
3 situation?

4 A. I assume so. It was probably bid
5 out the first time. After that, it's
6 probably been the same person.

7 Q. Okay. All right, I think that's
8 all. Thank you, sir.

9 A. You're welcome. Thank you. I
10 appreciate your courtesy.

11

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

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REPORTER'S CERTIFICATE

* * * * *

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

Kaley, Robert; Tolbert

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

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

11 This 12th day of May 2003.

12
13
14
15 _____
16 Angela Abbott Blankenship
17 Reporter and Notary Public
18 State of Alabama at Large

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