

COPY

Superior Court of the State of California
For the County of Los Angeles

TRANSWESTERN PIPELINE)
COMPANY,)

Plaintiff,)

vs.)

Case No. BC 026959

MONSANTO COMPANY and)
DOES 1 through 200, inclusive,)

Defendant.)

November 2, 1992

Deposition of GEORGE J. LEVINSKAS, taken on behalf
of Plaintiff.

GORE REPORTING COMPANY

Boatmen's Tower, Suite 1175 - 100 North Broadway
St. Louis, Missouri 63102
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1 Superior Court of the State of California
2 For the County of Los Angeles
3

4 TRANSWESTERN PIPELINE)

5 COMPANY,)

6 Plaintiff,)

7)

8 v.) No. BC 026959

9)

10 MONSANTO COMPANY and)

11 DOES 1 through 200,)

12 inclusive,)

13 Defendants.)

14

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17

18 Deposition of GEORGE J. LEVINSKAS,
19 taken on behalf of Plaintiff, at the offices
20 of Bryan Cave, One Metropolitan Square 500
21 North Broadway in the City of St. Louis,
22 State of Missouri, on the 2nd day of
23 November, 1992, before J. Bryan Jordan,
24 certified shorthand reporter and notary
25 public.

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APPEARANCES:

FOR THE PLAINTIFF:

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1 Whereupon. . .

2 GEORGE J. LEVINSKAS,
3 of sound mind, having been first duly sworn
4 to tell the truth, the whole truth, and
5 nothing but the truth in the case aforesaid,
6 testified upon his oath as follows, to-wit:

7 EXAMINATION

8 QUESTIONS BY MS. WELCH:

9 Q. Good morning, Mr. Levinskas.

10 A. Good morning.

11 Q. My name is Dana Welch. I
12 represent Transwestern Pipeline Company in
13 litigation that it has instituted against
14 Monsanto Company. Could you please state
15 your name for the record and spell it?

16 A. It's George J. Levinskas. That's
17 L-e-v as in Victor, -i-n-s-k-a-s.

18 Q. Have you ever been deposed, Dr.
19 Levinskas?

20 A. Yes, I have.

21 Q. In how many cases?

22 A. Perhaps a dozen or so.

23 Q. How many of those cases, if any,
24 related to polychlorinated biphenyls?

25 A. Probably half of them.

1 Q. Do you recall the names of any of
2 those cases?

3 A. Not particularly.

4 Q. When was the last time you were
5 deposed?

6 A. Probably about a year, year and a
7 half ago.

8 Q. Do you recall the name of that
9 case?

10 A. It was an acrylonitrile lawsuit.

11 Q. Excuse me?

12 A. Acrylonitrile.

13 Q. So that's not related to PCBs?

14 A. No, I don't recall the last -- no,
15 the last PCB deposition, I really don't
16 recall.

17 Q. You are probably familiar with
18 rules of depositions, but let me refresh your
19 recollection, if I may. The first and most
20 important rule for our court reporter, as he
21 will tell you, is please wait until I finish
22 a question and I will attempt to do the same
23 for you for answers. That's very important,
24 so the court reporter can get everything
25 down.

1 Another rule is that I'd like to
2 get your best recollection and answer to any
3 question that I pose, but if you don't
4 understand a question that I pose, please ask
5 me to rephrase it and I will do so. If you
6 answer a question, I will assume that you
7 understood my question, so anytime you need
8 rephrasing, please ask me to.

9 After the transcript is prepared,
10 you'll have a chance to review it and make
11 any corrections that you need to make, and --
12 off the record.

13 (Brief interruption.)

14 BY MS. WELCH:

15 Q. I should tell you that if you do
16 make any corrections, that I or another
17 attorney for Transwestern Pipeline Company
18 may make comments or may point that out to a
19 jury when this case does go to trial.

20 You understand today, sir, that
21 you are under oath, do you not?

22 A. Yes, I do.

23 Q. And so that means that even though
24 this is an informal setting in a conference
25 room, that it is just as if you are in a

1 court of law.

2 A. That is correct.

3 Q. All right. Could you please tell
4 me what your educational background is?

5 A. I have a Bachelor's degree in
6 chemistry and a doctorate in pharmacology.

7 Q. Where did you obtain your
8 Bachelor's degree from?

9 A. Wesleyan University.

10 Q. In what year?

11 A. 1949.

12 Q. And from what school did you
13 obtain your Ph.D.?

14 A. The University of Rochester.

15 Q. In what year?

16 A. 1953.

17 Q. When did you start working at
18 Monsanto Company?

19 A. July 1971.

20 Q. What was your job position at that
21 point?

22 A. I think the title was Product
23 Safety Manager or something of that sort.

24 Q. What were your job
25 responsibilities?

1 A. I was hired to formalize and
2 consolidate the environmental assessment
3 activities of the company and to centralize
4 them in the Medical Department.

5 Q. What do you mean by environmental,
6 environmental policies of the company?

7 A. I don't -- did I use the word
8 "policy"? The environmental -- what Monsanto
9 decided was that they should formalize the
10 environmental review to look at their
11 products, the kinds of questions that were
12 being raised about the products, and to
13 recommend actions that could be taken to
14 resolve the questions that were being raised.

15 Q. Do you know how that process was
16 conducted before you were hired?

17 A. It was done separately by various
18 people within the company. I do not have
19 details on it.

20 Q. So to the best of your
21 understanding, was it done by the various
22 product groups, as opposed to one person
23 within the Medical Department?

24 A. That's correct.

25 Q. So if there were a question about

1 a product in, for instance, the Functional
2 Fluids Group, there would be a person there
3 who would develop information on that
4 product, to the best of your understanding?

5 A. I would assume that there was a
6 person and that he would interact with others
7 in the company, including the Medical
8 Department, to decide what should be done.

9 Q. Was this a new position that was
10 created for you?

11 A. Yes.

12 Q. Did you ever become aware of why
13 Monsanto decided to create this position in
14 July of 1971?

15 A. Not specifically.

16 Q. How about generally?

17 A. I really, I really have no
18 background as to why or what initiated it.

19 Q. Who did you report to?

20 A. Mr. Elmer Wheeler was my immediate
21 supervisor.

22 Q. Did he report to Dr. Kelly?

23 A. He reported to Dr. Kelly, in turn,
24 yes.

25 Q. So you were within the auspices of

1 the Medical Department?

2 A. Yes.

3 Q. Were you responsible for all
4 products that the company was developing at
5 that point?

6 A. The initial purpose was to look at
7 new products and new uses of existing
8 products.

9 Q. And what, particularly, were you
10 supposed to look at in terms of new products
11 or new uses of existing products?

12 A. The operating units were to inform
13 the Medical Department either at an early
14 stage that they had a new product or they
15 were anticipating a new use of an existing
16 product. The physician then required a
17 review of this, some estimates of the
18 potential exposures from the use, review of
19 available information, and could include
20 recommendations for additional testing.

21 Q. Now, are you speaking about
22 toxicological testing?

23 A. Yes.

24 Q. Any other kind of testing?

25 A. If deemed necessary, it could be

1 recommendations for other types of testing.

2 Q. Were you to recommend the kinds of
3 testing to any particular product needed?

4 A. I'll go back and limit myself to
5 the new products and new uses, and I would be
6 one of the elements making recommendations
7 for such testing.

8 Q. Are you trained as a toxicologist?

9 A. My first employment was teaching
10 toxicology.

11 Q. Would you consider that your area
12 of expertise?

13 A. Yes.

14 Q. In July of 1971, when you came to
15 Monsanto, were there any other kinds of
16 testings -- testing besides toxicological
17 experiments that you were involved in
18 recommending?

19 A. The first several months was, was
20 getting just familiarized with what people
21 were doing, familiarity with the kinds of
22 products, and I really can't get more
23 specific than that. I don't recall.

24 Q. When, after you came, did you
25 become aware of PCBs?

1 A. Probably within a few months to
2 relatively soon.

3 Q. What percentage of your time in
4 1971 was spent working on the Product Group
5 that included PCBs or -- let me strike
6 that -- were spent working on PCBs?

7 A. I could not make an estimate, but
8 I would say very little.

9 Q. How did you become aware of PCBs?

10 A. Read about them in the scientific
11 literature, in the newspaper accounts, and
12 since Monsanto was a manufacturer of PCBs,
13 discussions by people within the company.

14 Q. When you first became aware of
15 PCBs when you came to the company, did anyone
16 tell you that Monsanto was engaged in any
17 kind of PCB program?

18 MR. PREUSS: Well, object as
19 ambiguous as to what do you mean by "PCB
20 program."

21 BY MS. WELCH:

22 Q. Go ahead and answer if you
23 understand.

24 A. Well, I would ask the same thing:
25 What, what's the program that you are

1 referring to?

2 Q. For instance, did anybody ever
3 tell you that there was a phased withdrawal
4 of PCB products?

5 A. No, at that time, I had no
6 knowledge of such.

7 Q. Did anyone ever tell you that
8 there were ongoing tests concerning the
9 effects of PCBs?

10 A. The effects in what systems?

11 Q. Any systems.

12 A. I don't recall specifically, I
13 don't recall anybody specifically telling me
14 that there were tests going on with PCBs, in
15 the sense that someone came up and said,
16 "Hey, here's something you should know about.
17 And as I said, gradual discussions, and so
18 forth, the information started coming out
19 that there were PCBs, and Monsanto was a
20 maker and Monsanto had an interest in those
21 products.

22 Q. Did you become aware in those
23 first few months that PCBs were being found
24 to be persistent in the environment?

25 A. Yes, that would have been

1 generally reported, and I probably knew that
2 before I came to Monsanto, but that was being
3 reported generally in the literature.

4 Q. Did anyone at Monsanto bring this
5 to your attention?

6 A. I cannot recall.

7 Q. When you say that it was generally
8 being reported in the literature, what
9 literature are you referring to?

10 A. Various scientific journals,
11 technical journals.

12 Q. Do you recall any of those right
13 now?

14 A. No, not specifically.

15 Q. Do you recall at the time that you
16 read these what journals you were subscribing
17 to that would have reported persistence of
18 PCBs in the environment?

19 A. I know the journals I was
20 subscribing to, but I also had access to
21 library resources where there were additional
22 journals, and it would be difficult to
23 pinpoint a journal or an article at this
24 time.

25 Q. At the time, were you reading a

1 scientific journal by the name of "Nature"?

2 A. Not very often, no.

3 Q. Do you read "Scientific American"
4 or did you at the time?

5 A. Not very often.

6 Q. Was there any reason before you
7 came to Monsanto that you had a particular
8 interest in PCBs?

9 A. I do not think I indicated a
10 specific interest in PCBs. I meant to convey
11 that in my general reading of scientific
12 literature, I became aware of PCBs, as I did
13 become aware of many other chemicals.

14 Q. When you came to Monsanto in July
15 1971, say, until the end of the year, did
16 anyone ever tell you that higher-chlorinated
17 biphenyls tended to persist as opposed to the
18 lower chlorinated biphenyls?

19 A. Again, I would have to say that
20 somewhere along the line, I became aware of
21 that, but I cannot specifically pinpoint when
22 or how.

23 Q. When you came to Monsanto, did you
24 do any reading to familiarize yourself with
25 issues of toxicity with respect to PCBs?

1 A. Not specifically.

2 Q. Did you do so with respect to
3 other chemicals that were being produced by
4 Monsanto?

5 A. My general recollection would be
6 that I was reading up or trying to find out
7 more information on the new products and the
8 new uses of products as they were brought to
9 my attention.

10 Q. Were there any new uses of PCBs
11 that were being considered at the time?

12 A. I cannot recall that there were.

13 Q. What products do you recall that
14 you were involved with at that point?

15 A. Predominantly, it would have been
16 agricultural products which Monsanto was
17 trying to bring to market.

18 Q. Were these chlorinated
19 hydrocarbons?

20 A. No.

21 Q. How long did you hold the position
22 of Product Safety Manager?

23 A. About a year or two.

24 Q. What was your next position?

25 A. It was Manager of Environmental

1 Assessment and Toxicology.

2 Q. Did your job responsibilities
3 change?

4 A. They had enlarged at that point,
5 and I had assumed responsibility for
6 toxicology.

7 Q. Who had responsibility for
8 toxicology prior to that time?

9 A. It was Dr. William Hunt.

10 Q. Did Monsanto, up to this point
11 when you assumed your new position, have
12 in-house capability for toxicology tests?

13 A. Not that I know of.

14 Q. Was there an outside laboratory
15 that was primarily contracted with for
16 toxicology tests by Monsanto?

17 A. All the testing was done at
18 several outside laboratories.

19 Q. Can you tell me the names of those
20 laboratories?

21 A. The predominant one was Industrial
22 Bio-Test. There were --

23 Q. Go ahead.

24 A. Shelanski Laboratories.

25 Q. Could you please spell that?

1 A. S-h-e-l-a-n-s-k-i. There were
2 contacts, work being done at several
3 universities, and there was a small
4 laboratory here in St. Louis, Younger
5 Laboratories.

6 Q. Where were the PCB toxicological
7 tests conducted, to your knowledge, up to
8 1972?

9 A. Industrial Bio-Test.

10 Q. When you say you assumed the
11 responsibility for toxicology, what did that
12 entail?

13 A. Along with the environmental
14 assessment which was being centralized in the
15 Medical Department, we were beginning to
16 centralize toxicity testing within the
17 Medical Department, so as we were making the
18 recommendations for possible testing, we
19 would then give them responsibility seeing
20 that that testing was carried out.

21 Q. I want to go back to what you mean
22 by environmental assessment with your first
23 job. Could you give me a description of what
24 environmental assessment meant in July 1971?

25 MR. PREUSS: I'm going to object

1 to mischaracterizing his testimony. I think
2 he said that environmental assessment was
3 part of his second job.

4 BY MS. WELCH:

5 Q. Was it also part of your first job
6 in 1971?

7 A. Can we go back to an earlier
8 question? I think I, I think I indicated
9 that it was to try to anticipate the kinds of
10 questions that might be arising, and that we
11 would recommend testing to see if we could
12 answer those questions.

13 Q. In terms of the impact of a
14 particular product on the environment?

15 A. It was not specifically
16 environment; it was to look at, if a product
17 was to be marketed, what kinds of questions
18 might logically or reasonably be raised by
19 people who would buy/use the product, and to
20 attempt to anticipate the kinds of questions
21 that would arise, assess those questions and
22 see if they needed some sort of testing to
23 provide information from which those
24 questions could be answered. So it would
25 include toxicity questions and it would,

1 could include some environmental aspects.

2 Q. Do you know how long Dr. William
3 Hunt had worked at Monsanto until 1972?

4 A. It was ten or twelve years,
5 something on that order.

6 Q. And was he responsible for
7 overseeing all toxicology?

8 A. He was the only toxicologist; I
9 would assume he was.

10 Q. Was he also responsible for
11 environmental assessment before you assumed
12 that position?

13 A. I can only indicate that my, my
14 position was intended to focus and centralize
15 these in the Medical Department. I'm not
16 aware of who or to what extent others
17 participated prior to that.

18 Q. Going back to the first job, did
19 you have contact with individuals within the
20 various product groups, as well as people in
21 the Medical Department?

22 A. Yes.

23 Q. So did you do some amount of
24 interfacing between the product groups and
25 the Medical Department?

1 A. Yes.

2 Q. Did you, in 1971, have contact
3 with the Functional Fluids Group?

4 A. Yes.

5 Q. Who in the Functional Fluids Group
6 did you have contact with at that point?

7 A. It would be difficult to recall
8 all the names because the Functional Fluids
9 Group was a large group with a variety of
10 products. John Herber was one, Bill Richard
11 was another; I don't know that O. P. Tanner
12 was in there or not. Ralph Munch. There may
13 be others, but I can't recall.

14 Q. How long did you stay in your
15 position as Manager of Environmental
16 Assessment and Toxicology?

17 A. In about '80 or '81.

18 Q. And what happened then?

19 A. I was named Director of
20 Environmental Assessment and Toxicology.

21 Q. How long did you remain in that
22 position?

23 A. Till about 1986.

24 Q. What happened then?

25 A. I was named Senior Toxicology

1 Consultant.

2 Q. How long did you remain in that
3 position?

4 A. September 1991.

5 Q. And what happened then?

6 A. I retired.

7 Q. Congratulations. Have you
8 consulted for Monsanto since that time?

9 A. This is my first contact with,
10 official contact with Monsanto since I
11 retired.

12 Q. Have there been any unofficial
13 contacts?

14 A. I go back to visit with my old
15 crew for lunch and that sort of thing, but
16 those were informal.

17 MS. WELCH: All right, I'd like to
18 introduce and have marked this exhibit as
19 Exhibit 1073.

20 (Plaintiff's Deposition
21 Exhibit 1073 marked for
22 identification.)

23 MS. WELCH: Exhibit 1073 is a
24 four-page document that bears the identifying
25 stamp of TRAN 013693 to TRAN 013696. It is

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1 entitled "A Listing of Toxicity and Related
2 Studies of Polychlorinated Biphenyls," and
3 I'd like to have you take a few minutes and
4 go over this document, if you could.

5 (Witness peruses said
6 document.)

7 A. Okay.

8 BY MS. WELCH:

9 Q. Have you ever seen this document
10 before, Mr. Levinskas?

11 A. I do not recall seeing this
12 document before.

13 Q. I'd like to go through the
14 listings of the reports and studies, and the
15 question I have to you with respect to each
16 one that is dated prior to or up through the
17 end of 1971 is, to the best of your
18 recollection, whether you recall examining
19 that report sometime in 1971 when you, after
20 you arrived at Monsanto.

21 First, the listing for the Harvard
22 School of Public Health report, do you recall
23 having examined that report?

24 MR. PREUSS: Is this at any time
25 after he joined Monsanto up through December

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1 31, 1971?

2 MS. WELCH: That's correct,
3 through -- July 1st, 1971, through December
4 31st, 1971.

5 A. I do not recall looking at those
6 reports during that time.

7 BY MS. WELCH:

8 Q. You mean the first report?

9 A. Right.

10 Q. Have you ever looked at that
11 report subsequent to December 31st, 1971?

12 A. I think those reports are, the
13 results of those reports are published in
14 some of the literature, and I can recall
15 reading about them at some later date. I do
16 not recall having looked at the specific
17 original report.

18 Q. Do you recall having read about
19 them prior to December 31st, 1971?

20 A. No, I do not think I read them
21 before December 1971.

22 Q. All right, the second one is the
23 Bernard Free Skin and Cancer Hospital. The
24 same question with respect to July 1st, 1971,
25 through December 31st, 1971.

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1 A. No.

2 Q. Have you ever read the report
3 subsequent to December 31st, 1971?

4 A. Yes.

5 Q. Do you recall approximately when
6 you read the report?

7 A. I would say probably in the latter
8 Seventies.

9 Q. Was there some kind of medical
10 library or any other kind of library that
11 contained reports of the various -- of
12 testing on various chemicals that Monsanto
13 produced?

14 A. Yes, there was a medical library.

15 Q. And that was in 1971?

16 A. Yes.

17 Q. Do you know whether that library
18 was divided into sections on products?

19 A. There was a, a trend, I don't know
20 that it was always followed, but there was a
21 trend to keep information either by products
22 or by laboratory.

23 Q. So if you wanted to go to the
24 library and read up on the reports to date on
25 PCBs, would those be easily accessible to

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1 you?

2 A. Well, at that time, I would have
3 had to ask somebody how to find my way in and
4 around the library. I would not have direct
5 access to the information. I mean, I
6 couldn't find my way around in the library to
7 access the information. I would have had to
8 ask someone for assistance.

9 Q. Why was that?

10 A. I wasn't familiar with how the
11 library was set up or organized.

12 Q. Do you know if these first-year
13 reports that we've looked at were in the
14 library in 1971?

15 A. I do not know that.

16 Q. Who maintained the library in
17 1971?

18 A. I, I do not specifically know. I
19 did not make that inquiry.

20 Q. If you were to want to go into the
21 library and find a particular product group
22 or laboratory, who would you ask?

23 MR. PREUSS: In 1971?

24 MS. WELCH: Yes.

25 A. I would have asked one of the

1 coworkers, Bill Hunt or Mr. Wheeler, what
2 they knew and where could I find out more
3 about it.

4 BY MS. WELCH:

5 Q. Number 3 is the Kettering
6 Laboratory reports, dated 1953 and 1955.
7 With respect to July 1st, 1971, to December
8 31st, 1971, do you recall having read this
9 report?

10 A. No, I do not.

11 Q. Do you recall having read
12 summaries of this report in any other
13 articles with respect to 1971?

14 A. The second one, yes, because --
15 the first one, I do not recall. I don't
16 recall if I ever read it.

17 Q. The second one or the first one?

18 A. I don't recall if I ever read the
19 first one. The second one I did read.

20 Q. Do you recall -- and was that in
21 1971?

22 A. No, that would have been much
23 later.

24 Q. Approximately when do you think
25 you read that report?

1 A. Mid to latter Seventies.

2 Q. Was there some reason that you
3 turned to this report and the other report
4 that you recalled seeing later in the mid to
5 late Seventies?

6 A. I can't recall a specific reason
7 for it.

8 Q. Did you develop some kind of
9 interest in, in looking at PCBs from a
10 professional standpoint at that point?

11 A. They were of increasing interest
12 and I, part of my keeping up-to-date, I did
13 more reading on them.

14 Q. And this was mid to late 1970's?

15 A. Probably that time.

16 Q. Is there any particular incident
17 or series of incidents that sparked your
18 interests at that point?

19 A. I cannot recall.

20 Q. With respect to the next,
21 Scientific Associates, there are two reports
22 that are listed there. Do you recall having
23 read those reports in 1971?

24 A. I don't recall ever reading those
25 reports.

1 Q. All right. The next lists a
2 report by Industrial Bio-Test Laboratories,
3 and you testified that Industrial Bio-Test
4 Laboratories was the outside lab that
5 conducted PCB tests for Monsanto. Were you
6 aware of this fact in 1971?

7 A. I was aware that studies were
8 underway or being finished up, yes.

9 Q. At Industrial Bio-Test?

10 A. At Industrial Bio-Test.

11 Q. The first report is subacute
12 dermal toxicity of Aroclor 1221, March 28th,
13 1963. During 1971, did you ever examine that
14 report?

15 A. I do not recall doing so.

16 Q. Did you ever look at that report
17 subsequent to 1971?

18 A. Yes, I believe I did.

19 Q. Do you recall approximately when?

20 A. Again, this would have been the
21 late, latter part of, mid to latter
22 Seventies.

23 Q. Number 2 is "Acute Toxicity
24 Studies with Aroclor." It says 122. That
25 might be 1221, might be a misprint, "Aroclor

1 5442 and MCS 1016," dated March 25th, 1971.

2 Did you review that report in 1971?

3 A. I do not recall that I did so.

4 Q. Do you recall having reviewed it
5 subsequently?

6 A. I really do not recall having
7 looked at that report.

8 Q. Number 3 is, "Toxicity, Mutagenic
9 Teratogenic" -- I'm probably mispronouncing
10 that -- "Reproduction and Residue Studies as
11 to Various Aroclors in Rats, Chickens, Mice
12 and Dogs," dated November 17th, 1971. Do you
13 recall having reviewed that report in 1971?

14 A. No, I do not.

15 Q. Is that the report that -- or the
16 studies that you testified were underway or
17 that you knew were underway when you arrived
18 at Monsanto?

19 A. They could be; I do not know.

20 Q. Do you recall ever having reviewed
21 that report later?

22 A. I do not recall having reviewed
23 that report, but --

24 Q. How about number 4, "90 days
25 Subacute Oral Toxicity Study with Aroclor

1 1221 in Beagle Dogs," dated December 30th,
2 1971; do you recall reviewing that report in
3 1971?

4 A. Not in 1971.

5 Q. Do you recall reviewing it
6 subsequently?

7 A. I may have but I don't recall.

8 Q. Okay, how about 5, 6 and 7? Those
9 are reports that are dated in 1972: Did you
10 review those reports at any time subsequent
11 to 1971?

12 A. I don't recall reviewing number 5.
13 I may have looked at number 6 somewhat later.
14 I am not sure about number 7, whether I did
15 or not.

16 Q. There are three reports that are
17 referred to under "Biological consultants."
18 One is an interim report for fish residue
19 analyses; the next is a report covering the
20 December 1970, March 1971, June 1971,
21 September 1971, for fish residue data, and
22 the other is the final progress report for
23 residue data. Did you review those at any
24 time?

25 A. I do not recall looking at any one

1 of those three reports at any time.

2 Q. All right, I'd like you to take a
3 look at the six reports that are referred to
4 under Younger Laboratories and tell me
5 whether you reviewed any of those reports at
6 any time in 1971.

7 A. I do not think I looked at any of
8 those in 1971.

9 Q. Did you look at any of them
10 subsequent to 1971?

11 A. I may have looked at the -- at 4,
12 5 and 6 at some later date. I, when I say I
13 may have looked at reports, if someone had
14 told me that the report was there and the
15 information, I would not have looked at the
16 report, or if someone said they were
17 uncertain, I may have gone back and looked at
18 the reports. That's why I have difficulty
19 saying whether I looked at the specific
20 report or not.

21 Q. Could you please explain your
22 answer a little bit better?

23 A. Well, if I asked someone and said
24 "Do we have a specific piece of information"
25 or a question, if he answered it, then I

1 would just leave it at that. If he said "Oh,
2 I think we have a report in the files," then
3 I would have gone and looked for the report,
4 and so, you know, specific items of
5 information, I can't always be sure just how
6 I got them, when I got them, or where I got
7 them.

8 Q. So if someone had said to you,
9 "This is the substance of the report,"
10 somebody on your staff, then you would have
11 relied on that?

12 A. Yes.

13 Q. That's what you are saying, as
14 opposed to just "The report exists, I don't
15 know what the substance is," then you would
16 have turned to the report?

17 A. That's correct.

18 Q. The last three are Monsanto
19 Industrial Chemicals Company's reports. Do
20 you recall having seen the first one in 1971?

21 A. No, I did not.

22 Q. Was Mr. Mees a colleague of yours?

23 A. Bill Mees was in the -- he was an
24 analytical chemist. I do not know what
25 specific unit of Monsanto he was assigned to.

1 Q. And he was not in the Medical
2 Department?

3 A. He was not in the Medical
4 Department.

5 Q. How about E. S. Tucker?

6 A. Scott Tucker was similar to Bill
7 Mees, a Monsanto employee but not in the
8 Medical Department.

9 Q. And how about W. J. Litschgi?

10 A. I do not recall the name Litschgi.
11 I don't know if I met him or not.

12 Q. With respect to the second report,
13 which relates to toxicity, determination of
14 polychlorinated biphenyl residues in beagle
15 dog tissues, dated December 1971, did you
16 review that at some point in your career?

17 A. I do not recall reviewing that at
18 all.

19 Q. How about the next one, which
20 refers to residues in white leghorn chickens?

21 A. Same response; I do not recall
22 looking at that report.

23 Q. At some point in 1971, did you
24 ever learn that the liver was the target
25 organ for PCBs?

1 MR. PREUSS: Object. Assuming
2 facts not in evidence.

3 BY MS. WELCH:

4 Q. You can answer.

5 A. Like all chlorinated hydrocarbons,
6 PCBs attack the liver. When and how I became
7 aware of this, I cannot recall.

8 Q. By "attack the liver," what do you
9 mean?

10 A. Well, they injure the liver at
11 very high doses.

12 Q. In 1971, did you ever become aware
13 that PCBs were an enzyme reducer?

14 A. I do not recall when I became
15 aware of that.

16 Q. Did you ever become aware that
17 PCBs are an enzyme inducer?

18 A. That's a well-accepted scientific
19 fact. I do not recall when I became aware of
20 it.

21 Q. Could you explain to me what
22 "enzyme inducer" means?

23 A. It produces an increase in the
24 enzyme activity of, in this case, the liver.
25 That's as general a description as I can give

1 you.

2 Q. And what effect does that have,
3 other than just the fact of increasing enzyme
4 production?

5 A. I'm not -- I guess I have to ask
6 what -- your definition of "effect," I guess
7 I don't understand. I need some
8 clarification of the question. I just don't
9 think I understand the question.

10 Q. Well, you said that it produces an
11 increase in the enzyme activity of the liver.

12 A. That's an observation, yes.

13 Q. All right, what effect does that
14 have when the enzyme activity of the liver is
15 increased? What happens?

16 A. The specific enzymes that they
17 want to look at in the liver tissue have a
18 greater activity than they normally do.

19 Q. Would you classify that as an
20 adverse effect?

21 A. I think it can be adverse or
22 beneficial.

23 Q. With respect to PCBs, it can be
24 beneficial?

25 A. I don't know which to expect in

1 the case of PCBs, but the effect could be
2 adverse or beneficial.

3 Q. Could you give me an example of
4 when an increase in enzyme activity of the
5 liver is beneficial?

6 A. The liver, among its many
7 functions, detoxifies chemicals, so if you
8 induce the activity of an enzyme that
9 detoxifies, diminishes the toxicity of a
10 harmful chemical, that would be a beneficial
11 effect.

12 Q. Is the reason that enzyme activity
13 is increased is that there is a toxic
14 chemical which is introduced in the case of
15 PCBs so the liver is, in effect, working
16 harder?

17 MR. PREUSS: Let me just ask for
18 clarification. Are you saying the only time
19 that you get increased liver enzyme is
20 because of a chemical? Is that the
21 implication of the question?

22 MS. WELCH: No, I'm talking, I'm
23 speaking with respect to PCBs and trying to
24 understand the process of why there is
25 induction of liver enzymes when a PCB is

1 introduced into an animal.

2 A. I think that as I said, a function
3 of the liver is to metabolize or detoxify,
4 attempt to modify foreign chemicals, so many
5 foreign chemicals, including PCBs, when they
6 are introduced into the animal body, the
7 liver responds as it should. It makes an
8 effort or increases its effort to eliminate
9 or somehow mitigate the effects of that corn
10 chemical, and that's, if the liver is doing
11 what it should be doing in an enhanced
12 fashion, I think that's a beneficial effect.

13 Q. By "beneficial effect," do you
14 mean that it, that the organism is, is helped
15 by that effect?

16 A. Yes, it detects a foreign chemical
17 and it's making an increased effort to get
18 rid of it.

19 Q. In the studies that you are aware
20 of with PCBs, have you observed any negative
21 effects from enzyme induction?

22 A. I don't, I don't recall, nor can I
23 decide how one would know or how one would
24 detect the negative effects from enzyme
25 induction in PCB studies.

1 MS. WELCH: I'd like to introduce
2 this as Exhibit 1074.

3 (Plaintiff's Deposition
4 Exhibit 1074 marked for
5 identification.)

6 MS. WELCH: Exhibit 1074 is a
7 two-page document that bears a number of
8 identifying marks. I'll use the one that's
9 on the back page, which is SCM 052788, and
10 it's dated October 13th, 1971. It appears to
11 be a memorandum from Dr. Levinskas to file:
12 Subject, Aroclor 1260, and could you please
13 take a minute to review this document.

14 (Witness peruses said
15 document.)

16 BY MS. WELCH:

17 Q. All right, Dr. Levinskas, are
18 these your initials and your handwriting on
19 the back page?

20 A. Yes.

21 Q. Is this a document that you
22 prepared?

23 A. Yes.

24 Q. Did you prepare it in the normal
25 course of business?

1 A. Yes.

2 Q. At or around October 13th, 1971?

3 A. Yes.

4 Q. And was it part of your regular
5 duties to prepare a document like this?

6 A. I frequently committed
7 conversations and items to documents such as
8 this. Whether or not it was considered part
9 of my duties or not, I don't know, but that
10 was my practice.

11 Q. Do you recall preparing this
12 document?

13 A. Yes.

14 Q. Do you recall the conversation
15 with Dr. Kimbrough?

16 A. Yes, in general.

17 Q. So you know who Dr. Kimbrough is?

18 A. Yes, I'd known her for several
19 years before I came to Monsanto.

20 Q. How did you know her?

21 A. I met her supervisor, Dr. Waylon
22 Hayes, in 1955, I believe, and then Dr. Hayes
23 had an interest in pesticides and when I was
24 at Cyanamid, working on pesticides, we
25 cemented our relationships; somewhere along

1 the line, he introduced me to Dr. Kimbrough.

2 Q. Do you regard Dr. Kimbrough as a
3 respected scientist?

4 A. Yes, I think she's very competent,
5 very capable.

6 Q. So you have a recollection of this
7 conversation with her?

8 A. In very general terms, yes.

9 Q. Do you recall anything other than
10 what's in this document about the
11 conversation with respect to Dr. Kimbrough's
12 finding of lesions on rat livers from
13 exposure to Aroclor? Or excuse me, it's
14 bladders. No, liver; strike that. First,
15 liver.

16 MR. PREUSS: Well, the question
17 is, does he recall anything about that
18 conversation, other than what's contained in
19 here relative to the issue of liver lesions?

20 MS. WELCH: That's correct.

21 A. My general recollection is that,
22 as I indicated earlier, like all chlorinated
23 hydrocarbons -- and there are pesticides,
24 many pesticides that are chlorinated
25 hydrocarbons -- this produced enlargement of

1 the liver, and she was proposing to present
2 those findings at the spring Society of
3 Toxicology meeting, which would have been in
4 the spring of '72.

5 Q. Did she contact you?

6 A. No, I called her.

7 Q. Do you recall why you called her?

8 A. Someone had indicated that they
9 thought she had found a bladder tumor, and it
10 was to inquire of her as to what information
11 she could provide me with respect to the
12 bladder tumors, and what I did then was
13 summarize the results of that phone
14 conversation for the record.

15 Q. Did she provide you with any
16 information about the bladder tumor?

17 A. Just beyond what's indicated in
18 the memo, no.

19 Q. Do you have any recollection of
20 any other discussion about the findings of
21 bladder tumor from exposure to Aroclor 1260?

22 A. Not with Dr. Kimbrough.

23 Q. With anyone else?

24 A. There was a meeting in the --
25 toward the end of '71, at a place called

1 Quail Roost, as I recall. I did not attend
2 the meeting, but several Monsanto people and
3 various other people were there, and I was
4 told that they had discussed this bladder
5 tumor and the conclusion, the consensus of
6 people that represented Monsanto, contract
7 lab, government regulatory agencies and
8 others, that the bladder was not related or
9 the bladder tumor was not related to the
10 feeding of the animals with the Aroclor.

11 Q. Was there -- did Monsanto have any
12 dispute with the findings of Dr. Kimbrough
13 with respect to liver lesions?

14 A. I do not recall that we, we did or
15 said anything about it.

16 Q. Were you surprised that she had
17 found liver lesions from exposure to Aroclor
18 1260?

19 A. No, as I've indicated, this is a
20 characteristic of chlorinated hydrocarbons.

21 Q. And you knew that at the time?

22 A. Yes.

23 Q. What is porphyria that's referred
24 to in the last paragraph?

25 A. It's an excretion of porphyrins in

1 the urine.

2 Q. And what are porphyrins?

3 A. Porphyrins are rather large
4 structures somewhat related, picture it
5 somewhat like the hemoglobin in blood, and
6 they appear in urine of animals, sometimes
7 spontaneously, sometimes as a result of
8 injury to tissues.

9 Q. And what does their appearance
10 indicate to the scientific observer?

11 A. Well, it would be, raise a
12 question for further -- to try to determine
13 where it was coming from, what was causing
14 it.

15 Q. In other words, it's not a normal
16 occurrence?

17 A. It may occur spontaneously, but
18 its incidence would be low.

19 Q. And what could cause porphyria?

20 A. A variety of things.

21 Q. Can you give me some examples?

22 A. I think what one will find
23 increased urinated porphyrins with lead
24 exposure, exposure to lead, for example.

25 Q. Was it a surprise to you to hear

1 that rats who had been exposed to Aroclor
2 1260 exhibited porphyria in their urine?

3 A. It did not particularly strike me.
4 I -- she did not give me sufficient details
5 about the degree of exposure, the duration of
6 exposure to make a rigorous assessment of
7 what she said.

8 Q. My question to you was, did it
9 surprise you?

10 A. I said not particularly.

11 Q. All right, on the second page,
12 there is a reference or there's a statement
13 made, here, "As a final note, Dr. Kimbrough
14 expressed concern over whether PCBs presented
15 an additional hazard to the employees who
16 manufacture it. I told her that because of
17 the chloracne and liver hazards, that there
18 had been medical supervision of the
19 employees. However, I would raise this point
20 with Dr. Kelly and Johnson for their review."

21 How did you become aware that
22 there had been medical supervision of the
23 employees because of the chloracne and liver
24 hazards?

25 A. I do not know that there were

1 specific measures taken on these employees.
2 That was a reference to the fact that we had
3 a Medical Department and physicians in
4 Monsanto's employ whose purpose was to assess
5 the health of employees, and I was intending
6 to convey, there, that all of our employees
7 were subject to medical supervision, and
8 therefore, I presumed whatever hazards people
9 might envision were being addressed.

10 Q. When did you become aware of the
11 effect of chloracne from exposure to PCBs?

12 A. I, I cannot specifically recall.

13 Q. But you did become aware of it at
14 some point in 1971?

15 A. I probably was aware of it before
16 I came to Monsanto.

17 Q. Did you have any discussions with
18 anyone at the Medical Department about
19 chloracne?

20 A. No.

21 Q. Did you have any discussions with
22 anyone in the Medical Department in 1971
23 about liver hazards with respect to PCBs?

24 A. No.

25 Q. Did you have any discussions with

1 anyone in the Medical Department about
2 particular attention being paid to chloracne
3 and liver hazards?

4 A. No.

5 Q. This was just based on your
6 general understanding of how the Medical
7 Department operated?

8 A. Yes.

9 Q. Did you raise the point with Dr.
10 Kelly and Johnson for their review?

11 A. The inclusion of them on this memo
12 was a means of bringing it to their
13 attention.

14 Q. Do you recall any discussions you
15 had with them subsequent to this memo about
16 this point?

17 A. I do not.

18 Q. Did the Medical Department,
19 including you, have regular meetings?

20 A. We did not have regularly
21 scheduled meetings in the sense that of a
22 fixed agenda or fixed calendar.

23 Q. Were there frequent meetings, even
24 if there weren't fixed agendas?

25 A. Our offices were in close

1 proximity and much of our discussion came up,
2 as subjects came up, they were discussed
3 without a formal meeting being called or
4 without everybody being included.

5 Q. So in the normal course of events,
6 would you have expected to discuss this
7 Kimbrough discussion with Dr. Kelly?

8 A. No, I would not have expected to.

9 Q. Why is that?

10 A. Dr. Kelly was my ultimate boss; I
11 brought the matter to his attention. If he
12 felt inclined to come discuss it with me, I
13 would listen or discuss it; I would not
14 approach him.

15 Secondly, we respect the
16 confidentiality of medical records, and I am
17 not a physician and I would not have
18 knowledge or access to the medical records,
19 nor would I expect to have knowledge or
20 access to the medical records.

21 Q. Did you bring to Mr. Wheeler's
22 attention the Kimbrough discussion, aside
23 from this memo?

24 A. He was my immediate supervisor, so
25 he should be informed as to what I'm doing,

1 yes.

2 Q. Do you recall any discussions you
3 had with him about this issue?

4 A. Oh, I suspect he came back and
5 asked me questions, but I don't recall
6 specifics.

7 MS. WELCH: I'm introducing
8 Exhibit 1075.

9 (Plaintiff's Deposition
10 Exhibit 1075 marked for
11 identification.)

12 MS. WELCH: Exhibit 1075 is a
13 multipage document with various identifying
14 stamps. I'm going to use SCM 058930 to SCM
15 058945, and it's entitled "Report Number M.S.
16 12002," dated October 14th, 1981, by Dr.
17 Levinskis and it is on Monsanto Technology
18 paper, form, and please take a few minutes to
19 review this document.

20 (Witness peruses said
21 document.)

22 (Witness peruses said
23 document.)

24 THE WITNESS: I think I'm familiar
25 enough with this. I may want to go back and

1 look at it again if you ask questions,
2 but --:

3 MS. WELCH: Oh, sure.

4 BY MS. WELCH:

5 Q. Dr. Levinskas, is this a report
6 that you authored?

7 A. Yes.

8 Q. And you recall authoring this
9 report?

10 A. Yes, I do.

11 Q. Did you prepare this report in the
12 normal course of business?

13 A. Yes.

14 Q. And it was part of your job
15 responsibility to prepare a report such as
16 this?

17 A. I considered it so.

18 Q. Did you author it on or about
19 October 14th, 1981?

20 A. Actually, I did it, just before
21 that. That's the date it was issued.

22 Q. How long did you work on this
23 report for?

24 A. It's hard to recall, but I could
25 have spent a couple of weeks checking data

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1 and references and putting it together.

2 Q. Now, you are aware that Monsanto
3 stopped producing PCBs in 1977, are you not?

4 A. Yes.

5 Q. Why were you preparing a report
6 some four years after the end of production
7 of PCBs?

8 A. PCBs were being increasingly
9 discussed by the world in general, and I have
10 indicated, I think, in the introduction,
11 here, that the Monsanto studies, while they
12 had been made available to regulatory
13 agencies, had -- there was no, really,
14 available point at which they could be
15 referenced, so I decided it would be useful
16 to pull together the information we had so
17 that in subsequent discussions, we could have
18 a source reference from whatever we knew
19 about -- whatever we knew, I say whatever
20 Monsanto, data Monsanto had developed with
21 respect to the possibility of PCBs in one
22 place, so it was pulling together to have a
23 reference and it was a tabulation, results of
24 those studies.

25 Q. Do you recall this as a complete

1 tabulation up to 1981 of the studies that had
2 been done?

3 A. No, this was --

4 MR. PREUSS: By Monsanto or -- he
5 said studies that had been done.

6 MS. WELCH: Studies had been done
7 in general.

8 A. No, he's indicating in the
9 abstract this is a tabulation of results of
10 microscopic evaluation of liver sections from
11 rats fed Aroclor 1242 and 1254 or 1260.
12 There is some general discussion leading into
13 it to point out that this information has
14 been distributed to various regulatory
15 agencies, but there was no single source
16 reference or summary that people could
17 access, and it was my hope that this could
18 provide such a single-source reference.

19 BY MR. WELCH:

20 Q. With respect to --

21 A. The three?

22 Q. The liver sections from rats fed
23 Aroclor 1242, Aroclor 1254 or Aroclor 1260
24 for two years?

25 A. That's correct.

1 Q. I'd like to turn to the
2 introduction which was on the page that's
3 enumerated number 1. There's a reference to
4 Monsanto sponsoring a series of animal tests
5 at Industrial Bio-Test in 1969. Are those
6 the tests that you referred to that you were
7 aware were underway in 1971 when you arrived?

8 A. Yes.

9 Q. And those tests consisted of
10 feeding studies to rats and dogs, one of the
11 tests?

12 A. Yes.

13 Q. Three-generation rat reproduction
14 study?

15 A. Yes.

16 Q. A rat teratology study?

17 A. Yes.

18 Q. What is teratology?

19 A. It's from the Greek word "terata,"
20 meaning monster. It's a study of birth
21 defects.

22 Q. A dominant lethal mutagenic study
23 in mice?

24 A. Yes.

25 Q. And what is lethal mutagenic

1 study?

2 A. That specific study involves
3 dosing male animals with a chemical to see if
4 it affects their sperm, and then mating those
5 animals with females to determine whether the
6 offspring will live or die. If there is a
7 dominant lethal defect, the pups will die, so
8 it measures the effects on the male sperm.

9 Q. And the last was a toxicity
10 reproduction study in chickens on each of the
11 three Aroclor products?

12 A. Yes.

13 Q. Do you recall if there were any
14 preliminary results that were issued when you
15 first got to Monsanto from these studies?

16 MR. PREUSS: Do you mean issued
17 before he started, or after the time he
18 started?

19 MS. WELCH: Well, whenever, did he
20 become aware of any preliminary results when
21 he arrived at Monsanto.

22 A. I did not -- well, I do not recall
23 being told of preliminary results in the
24 sense of results on ongoing studies. I
25 became aware that the studies were underway.

1 Mr. Wheeler was dealing predominantly with
2 polychlorinated biphenyl issues, and I had my
3 own assignments and I was not particularly
4 involved.

5 BY MS. WELCH:

6 Q. Did you become aware that, as this
7 document said, that the studies were
8 initiated by reports that PCBs had been
9 detected in the environment?

10 A. Yes, I was told that somewhat
11 later, or somewhere along the line. I don't
12 know just when.

13 Q. With respect to the protocols for
14 these, these experiments or these tests, were
15 they new protocols in 1969?

16 A. I do not know what the protocols
17 were when these studies were initiated
18 because I was not there at the time.

19 Q. Did you become aware of the
20 protocols when you reviewed the reports or if
21 you reviewed the reports?

22 A. I do not recall specific,
23 specifics about the protocols. That may or
24 may not have been there.

25 Q. Well, let me phrase it in this

1 way. Prior to 1969, were toxicologists
2 carrying out chronic feeding studies on rats
3 and dogs with respect to various other
4 chemicals, to your knowledge, besides PCBs?

5 A. Yes.

6 Q. Do you know in your background
7 when chronic feeding studies on rats and dogs
8 began to become a testing mechanism?

9 A. When I joined Monsanto in -- or
10 Cyanamid in 1958, there were two-year feeding
11 studies in progress, so with that as a
12 reference point, certainly in the 1950's, at
13 least.

14 Q. Okay, same question with respect
15 to three-generation rat reproduction study.

16 A. I would give a similar answer;
17 they were underway when I came to Cyanamid.

18 Q. That was in the mid Fifties?

19 A. I got there in 1958.

20 Q. How about with respect to a rat
21 teratology study?

22 A. Teratology studies did not
23 become -- terra -- let me start back. Some
24 aspects of teratology would be detected in a
25 reproduction study. What's labeled as a

1 teratology study, it really did not gain
2 prominence until the 1960's, with the
3 thalidomide tragedy.

4 Q. That was the early 1960's?

5 A. It would have been early 1960's.
6 It was during the Kennedy administration,
7 '62, '61, '2, '3.

8 Q. How about the dominant lethal
9 mutagenic study; when did that become
10 available?

11 A. Oh, I don't know when it became
12 available. It probably had been around a
13 long time. It was infrequently done, even in
14 the 1969 time frame, and sort of fell out of
15 favor, and it's probably getting a little bit
16 of a resurrection again.

17 Q. Was it available prior to 1969,
18 even if it was conducted infrequently?

19 A. Oh, I don't know the history of it
20 well enough, but the features of it certainly
21 were, were well -- known well before this,
22 yes.

23 Q. How about a toxicity reproduction
24 study in chickens; was that available prior
25 to 1969?

1 A. That would have been done, yes.

2 Q. In your experience, when were you
3 first aware of these kinds of studies with
4 respect to the toxicity reproduction studies
5 in chickens?

6 A. Outside of agricultural products,
7 I don't think they would have been done
8 particularly. It certainly was not a common,
9 a commonly done test in chickens unless there
10 was -- it was intended for either medication
11 of chickens or it was intended for poultry
12 treatments of some sort or it was a pesticide
13 product. Other than that, I doubt that
14 anybody was really doing it.

15 Q. How about with respect to either
16 pesticides or medicinal products for poultry?

17 A. That's what I said, veterinary or
18 things intended to treat chickens or
19 pesticides that could be a residue on crops
20 fed to chickens.

21 Q. Were those being done in the
22 1950's?

23 A. I don't -- I can't say. They were
24 not frequently done, is the best I could say,
25 because they were not being -- my earliest

1 chickens reproduction study that I would have
2 experienced would have been in the early
3 Sixties, by personal experience.

4 Q. Was this when you were at
5 Cyanamide (Phonetic)?

6 A. Yes.

7 Q. Am I pronouncing that right?

8 A. Sigh-AN-a-mid.

9 Q. Cyanamid. There's a reference in
10 the bottom paragraph on the first page,
11 "Starting in 1969, while these studies still
12 were in progress, information regarding them
13 was made available to various government
14 groups." Do you know what information
15 regarding them was made available to various
16 government groups? There is a reference to a
17 footnote.

18 A. My -- the basis for that comment
19 in my recollection is, in part, document
20 footnotes on page 8 that's information which
21 was in the files, and it is, my recollection
22 is that as interim reports or progress
23 reports, or whatever was being received by
24 Monsanto, it was being forwarded to the
25 regulatory personnel.

1 Q. Do you know whether the final
2 reports confirmed the interim reports or was
3 there a change in the results?

4 A. I did not compare interim to final
5 reports, but they have been widely
6 distributed. If there were inconsistencies
7 in the conclusions, I would expect that they
8 would have been brought to our attention.

9 Q. So by that, are you saying that
10 you don't think there were inconsistencies?

11 A. I do not think there were
12 inconsistencies, but I do not recall
13 comparing interim reports with final reports.

14 Q. How often do you know were interim
15 reports issued with respect to these tests?

16 A. I've indicated earlier, Mr.
17 Wheeler was dealing on the PCB issues, and I
18 really have no knowledge of that.

19 Q. I'd like to turn to page, the page
20 that's enumerated number 4 under "Methods."
21 What is a threshold limit value?

22 A. There is a group in the second
23 line, "ACGIH, the American Conference of
24 Governmental Industrial Hygienists, they set
25 standards of chemicals in the air that are

1 acceptable or, some say, permissible for
2 employee exposure. They revise that list
3 annually, they document the basis for those
4 numbers, and it is through that -- and this
5 1969, the second line refers to the latest
6 revision, but when I say I may have learned
7 of PCBs I have used the ACGIH threshold limit
8 value list many times, and I have read their
9 documentation, and I may well have gotten my
10 first exposures to PCBs by reading that
11 document in their documentation.

12 Q. Is there an encyclopedia or
13 dictionary of threshold limit values that's
14 issued by this group?

15 A. It's issued as a small pamphlet
16 annually.

17 Q. Was that available to people at
18 Monsanto?

19 A. Yes. It's available to everybody.

20 Q. Is it a respected reference book
21 for industrial hygienists?

22 A. Its membership consists of
23 industrial hygienists working for federal and
24 state governments, and I would have to
25 accept, assume and believe that it is highly

1 reputable.

2 Q. Is this a -- is this handbook --
3 was this handbook available in the medical
4 library at Monsanto?

5 A. Yes.

6 Q. And it was used by you and others
7 with reference to threshold limit values?

8 A. Yes.

9 Q. Now, there's a reference here in
10 the second line to the dosage that a person
11 would receive of PCBs, and then there is a
12 reference to dosages that rats would receive.
13 Why is there a reference under methods to the
14 dosage that a person would receive?

15 A. This is not a reference to a
16 dosage a person would receive. This is an
17 illustration of how one would go about
18 estimating the levels to which you could
19 expose animals, and so it's a calculation of
20 certain assumptions, and then taking those
21 assumptions from man over to the animal to
22 come up with some estimate as to what levels
23 would be tolerable by the animals.

24 Obviously, if the animals die on
25 the third day of exposure, it's very

1 difficult to conduct a two-year study.

2 Q. So, of course, toxicological
3 experiments are not conducted on human
4 beings; is that correct?

5 A. Not knowingly.

6 Q. Not intentional toxicological
7 experiments?

8 A. I'll say not knowingly. We hope
9 that we are -- we believe that we are not
10 conducting experiments on people.

11 Q. So the intent in performing animal
12 experiments is to draw parallels between the
13 exposure that a person would receive?

14 A. Well, it's illustrated the fact
15 that in reaching any conclusion, you want to
16 consider all the available information that
17 you have and see how you can come up with the
18 best possible conditions, and as I indicated,
19 to start several hundred or a thousand
20 animals on a test and find that one has to
21 abort the test because the animals have died
22 or something, that they don't tolerate it, so
23 one wants to minimize the false starts, and
24 one goes through these sorts of calculations
25 and assumptions to see whether there is a

1 consistency and whether it will serve to pick
2 a point on something that nobody really knows
3 about when you start the study.

4 Q. So the idea of a threshold limit
5 value is, you don't want to go over that
6 amount because you would kill the animal?

7 A. No, no, the threshold limit value
8 is a level that the American Conference of
9 Government Industrial Hygienists, an air
10 level that they said is acceptable for human
11 exposure in the workplace. So starting with
12 that, you say if that level is in the air, if
13 the person inhales, we know how much he
14 breathes in the course of a working day, and
15 if his lungs take everything out of the air
16 and keep it in the body, this is the amount
17 that that person will have gotten by the end
18 of the day, and we use that as a starting
19 calculation, but by no means is this the
20 level that people will get, because they are
21 not going to retain everything, the level may
22 be lower than the threshold limit value, and
23 so forth, but it's sort of calculating an
24 upper limit that one has a reasonable basis
25 for saying they should not get more than this

1 and they should be able to tolerate this.

2 Q. Is there any other reason for
3 drawing the parallel from people to rats, in
4 terms of dosage?

5 A. No, this is merely intended to
6 illustrate to people how -- the rationale for
7 picking the doses that were in the study.

8 I might add that Kimbrough's study
9 later on, others have used the same hundred
10 part per million is about as high as it'll go
11 in the rat without killing them.

12 Q. All right, turning to page 6,
13 under "Discussion," there is a statement,
14 here, that, "Since there were increased
15 incidences of vascular changes in the
16 cytoplasm of the" -- you'll have to help me.
17 You should perhaps read this because you know
18 these words --

19 A. "-- hepatocytes."

20 Q. " -- hepatocytes and focal
21 hypertrophy --"?

22 A. Hypertrophy.

23 Q. " -- at all dose levels for all 3
24 Aroclor products at the 24-month sacrifice, a
25 'no-effect' level was not established for

1 liver effects." Can you please explain to me
2 that conclusion?

3 A. That's evident from the data. A
4 no-effect level means that insofar as one can
5 tell through the measurement tools, no
6 effect, no, nothing was observed. Since
7 there was, this takes, let's take
8 specifically focal hypertrophy, which means
9 there are spots in the cell where there is an
10 increase in tissue, it's -- an old-timer once
11 used the analogy, if one goes out and chops
12 firewood for the winter, you will get muscle
13 hypertrophy. If you use muscles more, you
14 will get an increase in muscle mass. So if
15 the liver is doing -- we talked earlier
16 detoxification, it's got little spots, little
17 focal areas where there's an increase in cell
18 tissue, and that is an increase above
19 background, so there is an effect on the
20 liver. However, I don't think that could be
21 considered a deleterious effect, even though
22 there is an effect on the liver, so that's
23 the reason for saying a no-effect, in the
24 sense of nothing happening, was not
25 established for liver effects.

1 Q. Turning to the next page, page 7,
2 you -- there is a statement, here, that
3 "focal" -- pronounce the word again for me?

4 A. High-PER-truh-fee.

5 Q. "-- hypertrophy of the liver
6 occurred at 3, 3, and 12 months for rats fed
7 Aroclor 1260, 1254 and 1242 respectively, and
8 was not seen in livers of control animals."
9 By "control animals," you are referring to
10 animals who are not exposed to Aroclor.

11 A. Yes.

12 Q. So in other words, the result,
13 here, is that the Aroclor conclusively caused
14 the focal hypertrophy; is that correct?

15 A. This is, this is a restatement of
16 what we had talked about on the preceding
17 page or on page -- where was it? The
18 preceding page.

19 MR. PREUSS: That's the preceding
20 page.

21 A. Yeah, the preceding page, there
22 were subtle changes in the livers at all
23 three dose levels, so a no-effect level was
24 not observed. This is a restatement of what
25 it just discussed on the previous page.

1 BY MS. WELCH:

2 Q. The next statement is that,
3 "Livers of some animals fed 100 parts per
4 million of each Aroclor also had benign liver
5 tumors."

6 A. Yes.

7 Q. And then there is two kinds of
8 liver tumors, I assume, that are referred to.
9 Can you explain to me what the liver tumors
10 are that were seen at a hundred parts per
11 million?

12 A. A hepatoma is a benign tumor, not
13 malignant. A cholangiohepatoma is a, again,
14 a benign tumor in the bile duct. These
15 findings had been reported in the initial
16 two-year feeding studies, so there's no
17 change in --

18 Q. And those were the feeding studies
19 that began in 1969?

20 A. That's right.

21 Q. So this is a confirmation of those
22 studies, or is it a summary of those studies?

23 A. Well, let me see what -- give me
24 a --

25 (Witness peruses document.)

1 (Continuing) If you look at page
2 2, in the second paragraph, starting, the
3 two-year studies were done in that, in the
4 two-year studies consistent with general
5 practices, they looked at the livers from ten
6 animals in each group. That is, they
7 autopsied the animals, they'd look at the
8 livers and they would pick, if they saw
9 nothing unusual, they were instructed to look
10 at ten tissues from, or tissues from ten
11 animals in each group and they reported those
12 livers.

13 At a later date, when the question
14 came up as to whether they caused liver
15 cancer, we instructed the lab to go back and
16 look at all available livers, including
17 animals that had been on tests whose livers
18 had not previously been examined, and so this
19 report is looking at those to livers from
20 every available animal. In other words, the
21 ten that they looked at in the previous
22 studies and the ones that they had put in
23 formaldehyde and kept preserved, they looked
24 at all of them and when they looked at all of
25 them, basically they did not see any

1 differences from what they saw the first
2 time.

3 Q. So these were preserved from even
4 the earliest studies?

5 A. Right.

6 Q. And is there some kind of library
7 of slides or organs that Monsanto maintains
8 for past -- from past experience?

9 A. I do not know the current
10 retention policies, but yes, the wet tissues
11 are retained in formaldehyde and slides can
12 be retained, but they will deteriorate with
13 age, so I don't know the current retention
14 policy.

15 Q. For this report, the review was
16 made on everything from 1969 forward on the
17 liver tissue of rats?

18 A. The liver tissues from all the
19 rats in the three two-year feeding studies on
20 1242, 1254 and 1260 were examined, and this
21 summarizes the liver findings from all the
22 animals on those three two-year studies.

23 Q. Now, you refer to the question of
24 whether Aroclors cause liver cancer. How did
25 that question arise?

1 A. Dr. Kimbrough, we talked about
2 earlier, went back and started a two-year
3 feeding study --

4 MS. WELCH: Maybe we should go off
5 the record.

6 (Discussion off the record.)

7 BY MS. WELCH:

8 Q. Go ahead. I'm sorry, Doctor.

9 A. Dr. Kimbrough initiated,
10 unbeknownst to us, a two-year feeding study
11 with female rats to further explore the
12 question of the bladder cancer. She used
13 females because that's where she saw the
14 first bladder cancer she encountered and she
15 did only one high dose level.

16 When she -- it would have been
17 about '83, '4, I'm not sure; time's a blur --
18 she then called -- she then came to Monsanto.

19 MR. PREUSS: Did say '83 or '73?

20 THE WITNESS: I'm sorry, '73.
21 '72, '3, '4; somewhere in there, yeah.

22 A. (Continuing) She then called
23 Monsanto and came up to visit with us and
24 told us that she had found liver cancers in
25 the female rats fed high levels of Aroclor

1 1260.

2 As a result, to attempt to
3 determine, since we have two contradictory
4 pieces of information, our study which -- at
5 IBT which said there was no cancer and
6 Kimbrough's findings, we said, "We want you
7 to go back and look at all available livers
8 on the chance that you may have missed
9 something. We want to look at not just the
10 ten, but everything," and so this is the
11 result of all liver sections that were
12 reviewed.

13 BY MS. WELCH:

14 Q. Did Monsanto hire scientists to
15 review Dr. Kimbrough's findings, as well?

16 A. Yes.

17 Q. And what were the conclusions of
18 those scientists who reviewed her findings?

19 A. The -- there are two different
20 parts to this. The IBT, Industrial Bio-Test
21 pathologists went down and discussed their
22 findings with Kimbrough and they, they viewed
23 concurrently slides from, selected slides
24 from the Monsanto studies and selected slides
25 from Kimbrough's studies. Those conclusions

1 in general were that Monsanto did not have,
2 the Monsanto studies did not show liver
3 cancer. Some of the slides that Kimbrough
4 had, if they used a new classification that
5 Dr. Squires was developing, they would
6 consider cancers by Dr. Squires' definition.
7 We also went to the Epley Institute of Cancer
8 in Omaha, Nebraska with Dr. Schubic, who is a
9 cancer expert, and we asked what further
10 measures, what can we do in an attempt to
11 resolve the different findings, so he
12 recommended that we take Dr. Pour, Parvis
13 Poor, a pathologist, and he looked at all the
14 IBT slides and he looked at all of
15 Kimbrough's slides, and he did not feel that
16 Kimbrough's slides showed any evidence of
17 carcinogenicity.

18 Q. Was there any pathologist besides
19 Dr. Squires who reviewed Dr. Kimbrough's
20 slides?

21 MR. PREUSS: Well, I don't think
22 there's any testimony that Dr. Squires
23 reviewed them. It was Dr. Squires'
24 classification system.

25 MS. WELCH: I think he testified

1 that he reviewed both sets of slides.

2 MR. PREUSS: You misunderstood
3 him.

4 A. No.

5 MR. PREUSS:

6 MR. PREUSS: Dr. Pour, I think.

7 A. An IBT pathologist took slides
8 that were representative of the lesion that
9 is found, the hepatomas, the benign tumors,
10 and they met with, in Dr. Squires' office
11 with Dr. Kimbrough, and I think there was a
12 Dr. Levitt there. Squire and Levitt were the
13 originators of this new classification scheme
14 for liver tumors, and they looked at some
15 slides that this Dr. Kimbrough had brought, a
16 few slides that she thought illustrated her
17 points, and Industrial Bio-Test pathologists
18 did the same, so they looked at some slides,
19 but the only person who looked at all the
20 slides from all the studies was Dr. Pour.

21 Q. But the scientists who reviewed,
22 besides Dr. Pour, there were scientists who
23 agreed, looking at Dr. Kimbrough's slides,
24 that what she had found was, indeed, cancer?

25 MR. PREUSS: I'll object. That

1 mischaracterizes what he said.

2 A. I think I, as far as I know, and I
3 think he said, is that at that meeting, Dr.
4 Squire and Dr. Levitt, using their
5 classification scheme, characterized those
6 liver lesions, some of those liver lesions,
7 as cancerous.

8 BY MS. WELCH:

9 Q. Now, did Dr., up to the time that
10 you left Monsanto in 1991, did you maintain
11 contact with Dr. Kimbrough?

12 A. I had informal contact with her
13 many times subsequent to this, yes, but I
14 can't --

15 Q. Did Dr. Kimbrough, to your
16 knowledge, ever retract the findings of her
17 study or did she maintain that she had been
18 correct in her studies?

19 A. Well, she never -- she published
20 her data, and I don't know that there's ever
21 been a retracton, or -- she's never come up
22 to me and said, "I'm going to take it all
23 back, publish it," no.

24 Q. As far as you know, did she still
25 uphold the results of her studies?

1 A. I think it's interesting, she
2 wrote a review sometime later on the effects
3 of polychlorinated and polybrominated
4 biphenyls, and at the end of it, she says,
5 despite the interesting effects in animals,
6 there is no indication that they have caused
7 harm to man at the levels to which the
8 population is exposed.

9 Q. Was she speaking specifically of
10 carcinogenic harm?

11 A. She's talking about the spectrum
12 of biological effects, including
13 carcinogenicity.

14 Q. And what article is this?

15 A. An annual review of pharmacology
16 and toxicology, oh, five years ago or so. I
17 don't have the specific reference.

18 Q. With reference to her particular
19 findings, finding cancers in rats, that was
20 the question, did she ever, to your
21 knowledge, or does she still, to your
22 knowledge, uphold those findings, not with
23 reference to man but with reference to her
24 findings in rats?

25 A. I, I mean she's presented her

1 findings to us and she's published them, and
2 I, you know, I really haven't specifically
3 discussed them with her to see whether she
4 upholds them or denounces them.

5 MS. WELCH: Okay, why don't we
6 take a break.

7 (Recess)

8 MS. WELCH: All right, back on the
9 record.

10 BY MS. WELCH:

11 Q. I'd like to introduce as Exhibit
12 0176 the following document.

13 (Plaintiff's Deposition
14 Exhibit 1076 marked for
15 identification.)

16 BY MS. WELCH:

17 Q. Exhibit 1076 is a multipage
18 document that has a number of identifying
19 stamps. I will use the one SCM 019639
20 through SCM 019645. It's entitled "Toxicity
21 and Environmental Effects of Commercial
22 PCBs," and I note that on, the page stamped
23 SCM 019643 there appear to be the initials
24 "GJL." Please take a few minutes to review
25 this document.

1 (Witness peruses said
2 document.)

3 A. All right.

4 BY MS. WELCH:

5 Q. All right, turning to the page
6 that is marked SCM 019643, is that your
7 handwritten, are those your handwritten
8 initials?

9 A. No, they are not.

10 Q. Do you know whose initials those
11 are?

12 A. Well, they're my initials, but I
13 don't know who put them there.

14 Q. Did you author this document?

15 A. I believe I did. It sounds like,
16 reads like what I would have written.

17 Q. Do you recall authoring this
18 document?

19 A. Not specifically.

20 Q. But you believe that you did?

21 A. Yes. This is the sort of summary
22 I would be inclined to prepare, or the way I
23 would write it.

24 Q. And did you author this kind of
25 document in the normal course of your

1 business activities?

2 A. I would frequently summarize what
3 was going on with the product, yes.

4 Q. It was part of your job
5 responsibilities to do so?

6 A. Yes.

7 Q. Do you have any recollection of
8 the approximate time period that you authored
9 this document?

10 A. No.

11 Q. All right, turning to the section
12 that is entitled "Conclusions Based on
13 Monsanto Studies," the second part refers to
14 threshold limit values. That is the same as
15 in the document we discussed previously,
16 that's the same definition?

17 A. Yes.

18 Q. The next statement is, "The
19 no-effect level for these materials in
20 chronic rat and dog feeding studies is about
21 10 parts per million in the diet." Does that
22 mean that any amount above that causes some
23 kind of effect?

24 A. Yes, there would be observable
25 effects above that.

1 Q. And when you said that no
2 hepatocellular carcinomas were present, you
3 mean that there were no, there was no
4 evidence of liver cancer?

5 A. That's correct.

6 Q. In dogs and rats in that feeding
7 study.

8 A. Right.

9 Q. The next statement is, "The
10 no-effect level on rat reproduction is
11 between 1 and 10 parts per million," and you
12 state, "Since higher levels result in low
13 mating indices, that means that above 10
14 parts per million, the incidence of mating
15 decreases"?

16 A. Yes.

17 Q. And no teratogenic or mutagenic
18 effects were observed in studies with rats
19 and mice; is that correct?

20 A. Yes.

21 Q. The next statement is that "In
22 chicken reproduction and teratology studies,
23 Aroclor 1242, which produced the most severe
24 effects, had a no-effect level of 2 to 4
25 parts per million in the diet." Does that

1 mean that above 4 parts per million, there
2 was an effect?

3 A. There were differences from the
4 controls that were statistically significant.

5 Q. And again, the controls were
6 chickens that were not fed any Aroclor?

7 A. That's right.

8 Q. The next statement is that "Acute
9 toxicity to fish varies from less than one
10 part per million to greater than 100 parts
11 per million depending upon the specific
12 Aroclors and species of fish tested." Is
13 that correct?

14 A. Yes.

15 Q. So there's a wide variety of
16 effects depending on the species and the type
17 of Aroclor?

18 A. Yes.

19 Q. Do you know what is acute toxicity
20 mean?

21 A. It's an index or a measure of
22 either a single dose or several doses given
23 over a short period of time with respect to
24 the -- most generally, it's a measure of the
25 acute lethality of the compound; the death.

1 Q. So what you are referring to,
2 here, is variety in dosage with respect to
3 death in the fish?

4 A. Yes, I believe that's what it is.

5 Q. The last statement is, "PCBs
6 containing three or less chlorine substitutes
7 per molecule are reasonably biodegradable."
8 Is that what your finding was?

9 A. I was told that by others.

10 Q. Do you know who told you that?

11 A. Not specifically.

12 Q. That was an assumption that you
13 based your work on, however?

14 A. I don't recall who told me or
15 when, but it was based on studies that
16 Monsanto had conducted.

17 Q. The next section is entitled
18 "Summaries of Monsanto's Long-Term Toxicity
19 Studies On Commercial PCBs." Was the
20 reference to the Industrial Bio-Test 1969 to
21 1971 tests?

22 A. The two-year lifetime, yes, these
23 would be the studies at Industrial Bio-Test.

24 Q. That started in 1969?

25 A. Somewhere about that there.

1 Q. In the earlier document, we saw a
2 reference to tests that began in 1969, and
3 I'm wondering whether these are the same
4 tests that --

5 A. These would be the same tests.

6 Q. There's a summary of the effect on
7 rats in the lifetime rat feeding studies.
8 Could you please -- and that's in that second
9 paragraph. Could you please summarize those
10 results?

11 MR. PREUSS: Counsel, you've got
12 to give us a better reference, please.

13 BY MS. WELCH:

14 Q. All right, it's under "Summary of
15 Monsanto's Long-Term Toxicity Studies On
16 Commercial PCBs"?

17 MR. PREUSS: Right.

18 MS. WELCH: The second paragraph
19 begins, "The highest dietary level" --

20 MR. PREUSS: All right.

21 MS. WELCH: -- and I'm just asking
22 the doctor to summarize the results from
23 those Industrial Bio-Test tests with respect
24 to lifetime rat feeding studies.

25 MR. PREUSS: You want him just to

1 read it?

2 MS. WELCH: Well, if he can
3 compress it, compress the findings in a few
4 sentences, that would be helpful.

5 MR. PREUSS: Well, I would say
6 that the document speaks for itself.

7 A. I've attempted to summarize
8 two-year studies, and I don't -- I attempted
9 to include everything I thought was relevant
10 for someone to know, and condensing it
11 further without going back and looking at the
12 other studies, I think, would be almost an
13 exercise in futility.

14 BY MS. WELCH:

15 Q. All right, let's go through it.
16 The statement, here, is that "The highest
17 dietary level of 100 parts per million
18 produced a weight depression after 24 months
19 in females fed Aroclor 1254." Is that
20 correct?

21 MR. PREUSS: Female rats.

22 MS. WELCH: Well, it just says "in
23 females," but the reference, of course, is to
24 rats.

25 MR. PREUSS: Lifetime rat feeding

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1 studies.

2 MS. WELCH: We understand it's
3 rats.

4 MR. PREUSS: Let's make sure the
5 record does.

6 MS. WELCH: The record should
7 reflect it refers to rats. We already know
8 human beings were not intentionally fed
9 Aroclor 1254.

10 BY MS. WELCH:

11 Q. Is that correct?

12 A. I would have to say, based on what
13 I've written here, that that is correct.

14 Q. So that would be a weight
15 compression of the overall body weight that
16 you are referring to?

17 A. Yes.

18 Q. And that is a liver weight
19 increase in all groups except males fed
20 Aroclor 1242; is that correct?

21 A. Yes.

22 Q. So that's referring to the three
23 Aroclors that were fed, 1254, 1242 and 1260?

24 A. With respect to these studies, in
25 body weight and liver weights are

1 specifically referred to as Aroclor 1254 and
2 1242, but the 1260 did not have those
3 effects.

4 Q. So there were liver weight
5 increases in all groups except males fed
6 Aroclor 1242? Is that correct?

7 A. Yes.

8 Q. And the next statement is that
9 "The important histopathologic changes were
10 present in the livers of animals sacrificed
11 at the end of the study." What is a
12 histopathologic change?

13 A. The tissue is, a thin slice of
14 tissue is put under a microscope, stained, to
15 highlight various tissues within or
16 structures within the tissue, and then it's
17 looked at microscopically. In the judgment
18 of the pathologists who did the studies, the
19 important or the significant changes, they
20 felt, were confined to the livers.

21 Q. And those are reflected in the
22 next sentence, where it states that it's
23 focal hypertrophy cytoplasmic lipid changes,
24 and in some animals from the 100 parts per
25 million group, hepatomas or

1 cholangiohepatomas; is that correct?

2 A. Yes.

3 Q. And I believe that we defined all
4 of those earlier except for cytoplasmic lipid
5 changes. What are those?

6 A. The cytoplasm is the fluid part of
7 a cell, and lipid would be substructures or
8 portions in that fluid that take up a fat
9 stain, so some changes in fatty content, of a
10 fatty nature within the cells.

11 Q. So there was impact on the liver
12 in the animals sacrificed, but there was no
13 evidence of hepatocellular carcinogenicity of
14 the Aroclors. Is that correct?

15 A. Yes.

16 Q. In terms of dogs, there were some
17 slight decreases in body weight gain? Is
18 that correct?

19 A. Yes.

20 Q. And the next statement is, "At the
21 highest feeding level of a hundred parts per
22 million, Aroclor 1260 produced an increase in
23 serum alkaline phosphatase activity and liver
24 weights without concomitant histopathological
25 changes." Can you please explain to me what

1 that sentence means?

2 A. At a hundred ppm of Aroclor 1260,
3 there were no microscopic exchanges in the
4 appearance of the tissue. However, there was
5 an increase in serum alkaline phosphatase
6 activity, which is an enzyme in the blood,
7 and there was an increase in the weight of
8 the livers.

9 Q. Would this be an example of enzyme
10 induction?

11 A. Serum alkaline phosphatase is --
12 I'd have to go back and check specifically
13 which one, but would more likely be a rupture
14 of or a breakdown of a cell or somehow leaked
15 out of a cell. It would not -- I would not
16 consider it an induction of enzyme activity.

17 Q. In addition to those changes that
18 were noted, there were evidence of
19 gastrointestinal inflammatory lesions and
20 ulcerations? Is that correct?

21 A. Yes.

22 Q. And those were similar to those
23 found by an experimenter by the name of Allen
24 in rhesus monkeys fed Aroclor 1248?

25 A. Yes.

1 Q. The next reference in the
2 following paragraph is to rat reproduction
3 studies, and the effect that was found here
4 was that in the second and third generations,
5 there was a reduction in mating index at the
6 two highest feeding levels, which were 10
7 parts per million, a hundred parts per
8 million. Is that correct?

9 A. Yes.

10 Q. No other changes were noted in rat
11 reproduction studies?

12 A. That's right.

13 Q. The next statement is that there
14 was no evidence of teratogenic or mutagenic
15 changes in rats and mice?

16 A. For the studies that were
17 conducted, there were no changes observed.

18 Q. With reference to the chicken
19 toxicity reproduction study, there was a
20 statement earlier that Aroclor 1242 had the
21 most severe effect with respect to toxicity
22 and reproduction. Do you recall that? It's
23 on page -- the second full page.

24 A. Yes.

25 Q. And what was the effect of the

1 Aroclor?

2 A. There was a decreased egg
3 hatchability.

4 Q. What does that mean?

5 A. The eggs are taken from the hens
6 and they're put in an incubator and determine
7 how many, which ones hatch.

8 Q. Was there a decrease in eggshell
9 thickness as the result of the Aroclor
10 exposure?

11 A. Yes, there was a reduction in the
12 eggshell thickness.

13 Q. Any other effects?

14 A. It says chick viability was
15 affected by both substances at a dietary
16 level of 10 ppm for 1242.

17 The young chicks did not survive
18 as well.

19 Q. And was this different for the
20 different Aroclors? In other words, 1242 had
21 a more severe effect than the 1254 or 1260?

22 A. Yes.

23 Q. All right, the statement in the
24 next paragraph refers to biodegradation
25 studies. The statement is made that higher

1 chlorine-containing members are more
2 resistant to biodegradation and they
3 accumulate more readily and are less readily
4 metabolized and/or excreted from lipoid
5 tissue in animals. Does lipoid tissue refer
6 to fatty tissue?

7 A. Yes.

8 Q. Is fatty tissue where the PCBs are
9 accumulated?

10 A. Yes, similar to other chlorinated
11 hydrocarbons.

12 Q. So what you would expect to find
13 would be accumulation of the higher
14 chlorinated biphenyls and metabolism of the
15 lower chlorinated biphenyls. Is that right?

16 A. One would anticipate that as a
17 possibility.

18 MS. WELCH: Let me introduce the
19 following document as Exhibit 1077.

20 (Plaintiff's Deposition
21 Exhibit 1077 marked for
22 identification.)

23 MS. WELCH: Exhibit 1077 is a
24 three-page document bearing the identifying
25 stamp of TRAN 022439 to TRAN 022441. It is

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1 dated January 31st, 1972 and it is entitled
2 "Progress Report, Organic Chemical Division."
3 The project title is "Environmental
4 Analytical Program Toxicology Support
5 Studies," and it's reported by a number of
6 persons. The distribution includes a list
7 that includes Dr. Levinskas. Please take a
8 few minutes to review this document.

9 (Witness peruses said
10 document.)

11 MR. PREUSS: I think, just to be
12 accurate, it says, "Distribution to E. P.
13 Wheeler/G. Levinskas."

14 (Witness peruses said
15 document.)

16 THE WITNESS: All right.

17 BY MS. WELCH:

18 Q. Dr. Levinskas, do you recognize
19 this document?

20 A. I do not recall seeing this
21 document before.

22 Q. Is this the kind of document that
23 you would have received in your job in 1971
24 or 1972?

25 A. I could have received some, yes.

1 Q. Were you aware that the persons
2 who were listed as reporting this document
3 were conducting studies of the selected
4 chicken tissues in eggs from the Industrial
5 Bio-Test reports or studies?

6 A. I do not recall, I do not recall
7 what they were doing for analyses.

8 Q. Can you tell me what a PCB lipid
9 storage level is?

10 A. I can read the statement there.
11 It's not a standard technical term, so I
12 can't give you a formal definition for it.

13 Q. Do you know why just an
14 interpretation of the table, what the
15 reference is to one part per million, ten
16 part per million, 100 part per million? Is
17 that the amount that the animal is exposed
18 to?

19 A. That would be the -- that would
20 correspond with the levels in the diets that
21 were fed to the animals.

22 Q. So in other words, this would,
23 under one part per million, the analysis of
24 the tissue would be there would be a finding
25 of 28 parts per million that was retained by

1 the tissue? For Aroclor 1242, as an example?

2 A. Yes, that would be 28 parts per
3 million in lipids for 1242.

4 Q. And similarly, for the animal that
5 was fed 10 parts per million, there would be
6 196 parts per million in the lipid tissue?

7 A. Yes.

8 Q. And similarly, for a hundred parts
9 per million, there would be 2,356 parts per
10 million?

11 A. Yes.

12 Q. And so on.

13 Turning to page number 2, the
14 fourth paragraph, the last line of the fourth
15 paragraph states, "Extensive alterations of
16 the homolog distributions of those less
17 chlorinated products are being observed on
18 the EC/GC chromatograms." Were you aware of
19 this fact at around the time?

20 A. I've indicated I have not seen
21 this number before. There is a reference to
22 analysis, and I have no knowledge of it.

23 Q. You have no separate knowledge of
24 how the chromatograms looked when an animal
25 or a chicken had metabolized the PCBs?

1 A. No.

2 MS. WELCH: I introduce this as
3 Exhibit 1078.

4 (Plaintiff's Deposition
5 Exhibit 1078 marked for
6 identification.)

7 MR. PREUSS: Just to reference,
8 that last paragraph referred to dogs not
9 chickens.

10 MS. WELCH: You are right.

11 BY MS. WELCH:

12 Q. Do you have any knowledge of the
13 analysis of dog tissue?

14 A. I'm equally ignorant about dogs
15 and chickens.

16 BY MS. WELCH:

17 Q. Exhibit 1078 is a two-page
18 document bearing the identifying stamp of
19 TRAN 022437 to TRAN 022438. It is dated
20 November 30th, 1971, it's a report by various
21 persons, distribution to, amongst others, Mr.
22 Wheeler and Dr. Levinskas, and it is entitled
23 "Environmental Analytical Program --
24 Toxicology Support Studies."

25 Please take a few minutes to

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1 review this document.

2 (Witness peruses said
3 document.)

4 BY MS. WELCH:

5 Q. Have you ever seen this document
6 before?

7 A. I do not recall having seen this
8 document before.

9 Q. Do you know what the toxicology
10 support studies were?

11 A. No, I do not know what that -- I'm
12 not aware of the meaning of that term in this
13 connection.

14 Q. How about the environmental
15 analytical program?

16 A. Again, I have not seen the report.
17 I have no, no knowledge as to what was
18 intended.

19 Q. Were you aware generally of an
20 environmental analytical program that was
21 going on in November 1971?

22 A. I knew that they had chemists who
23 were doing analyses. I have no knowledge as
24 to whether, whether the formal, informal
25 program or the details of such analysis.

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1 Q. With reference to the chart on the
2 bottom with PCB lipid storage levels, is your
3 testimony that I could read the chart the
4 same way as I could from the previous
5 document, which is the feeding level is one
6 part per million and the result is what is
7 found in the tissues?

8 A. I would say that the, the two are
9 analogous.

10 Q. And this refers to the beagle
11 dogs?

12 A. This refers to dogs. The other
13 was rats.

14 Q. I believe the other was chickens.

15 A. Laboratory animals. Oh, it was
16 chickens, okay. Chickens.

17 Q. The statement is made on the
18 second page, "Alterations of the homolog
19 distributions were observed with all of the
20 products fed and were much more extensive
21 than previously noted in any of the other
22 tissue residue studies." Were you aware of
23 this fact with reference to dogs?

24 A. This is -- no, I was not aware of
25 this, as I was not aware of the earlier,

1 somewhat similar comment.

2 Q. Is there a reason in why the
3 species of chickens, rats and dogs were
4 chosen for the two-year study?

5 A. I do not know why those were
6 chosen.

7 Q. Is it fairly typical to experiment
8 on these three species with respect to
9 effects of chemicals?

10 A. I think we indicated earlier in
11 discussing the kinds of tests, certainly
12 rodent studies were very common. Dog studies
13 are fairly frequent, but less common, and
14 with the exception of veterinary
15 pharmaceuticals for poultry or possibly
16 pesticides, chicken reproduction studies
17 would be the least common.

18 Q. Are dog studies included because
19 it represents a higher form of mammal life
20 than a rodent?

21 A. The rationale is to use different
22 species. If there is a similarity in
23 response between different species, one will
24 have a greater confidence that a human
25 response may be similar, may be more general

1 biological. If there are wide variations or
2 differences between rats and dogs, then the
3 prudent assumption is that the most sensitive
4 species may well be indicative of man, but
5 that's an assumption that is made almost
6 automatically.

7 Q. So reflecting what you said, the
8 assumption would be that the species that
9 responds with the most adverse effects, the
10 assumption would be that's how man would
11 respond?

12 A. Correct, unless one had
13 information indicating that that particular
14 animal species was not necessarily
15 representative.

16 Q. And here, we're talking about
17 studies amongst mammals or are we talking
18 about studies amongst any species?

19 A. Ideally, one would like mammalian
20 data, but if one only had chicken data, one
21 would use chicken data in a similar manner.

22 MS. WELCH: I would introduce this
23 as Exhibit 1079.

24 (Plaintiff's Deposition

25 Exhibit 1079 marked for

1 identification.)

2 MS. WELCH: Exhibit 1079 is a
3 multipage document. It's a progress report
4 from the Organic Chemicals Division dated
5 October 29th, 1971, entitled "Environmental
6 Analytical Studies Research and Development
7 Manufacturing and Outside Support Studies."
8 The distribution includes Mr. Wheeler and Dr.
9 Levinskas. The identifying stamp is TRAN
10 022427 to TRAN 022436.

11 (Witness peruses said
12 document.)

13 BY MS. WELCH:

14 Q. Do you recall having seen this
15 document before?

16 A. I do not recall having seen it.

17 Q. There is a distribution list,
18 here, which you are included on and there is
19 an asterisk next to your name, as well as to
20 Mr. Wheeler's name and the statement is above
21 that, the asterisk refers to "with details,"
22 and then the document contains numbered pages
23 that are called details. Do you recall
24 having seen any of these details in the back?

25 A. I do not recall.

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1 Q. Turning to page 6, "Customer
2 Service, General Motors Corporation, Buick
3 Division," reviewing this section, do you
4 have any recollection of analyzing PCB
5 releases or PCB samples from Buick's plant
6 effluent in nearby drainage streams?

7 A. I have never analyzed any PCB
8 effluents.

9 Q. Did you ever hear of that having
10 been done by others?

11 A. I do not recall hearing of it.

12 Q. Do you recall hearing of any
13 pressure from the Michigan State Regulatory
14 Agency?

15 A. I do not recall hearing any
16 pressure from the state regulatory agency,
17 no.

18 MS. WELCH: All right, I don't
19 have any further questions.

20 MR. PREUSS: I have Mr. Papageorge
21 scheduled for tomorrow at 9:00. Is that
22 okay?

23 MS. WELCH: Yes.

24 (Whereupon, the deposition
25 concluded at 11:35 a.m.)

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COMES NOW THE WITNESS, GEORGE
J. LEVINSKAS, and having read the foregoing
transcript of the deposition taken on the 2nd
day of November, 1992, acknowledges by
signature hereto that it is a true and
accurate transcript of the testimony given on
the date hereinabove mentioned.

GEORGE J. LEVINSKAS

Subscribed and sworn to before me
this _____ day of _____, 1992.

My Commission expires: -----

Notary Public

1 STATE OF MISSOURI)

2 SS:)

3 CITY OF ST. LOUIS)

4 I J. Bryan Jordan, notary public
5 in and for the State of Missouri, duly
6 commissioned, qualified and authorized to
7 administer oaths and to certify depositions,
8 do hereby certify that pursuant to agreement
9 in the civil cause now pending and
10 undetermined in the Superior Court of the
11 State of California, to be used in the trial
12 of said cause in said court, I was attended
13 at the offices of Bryan Cave, in the City of
14 St. Louis, State of Missouri, by the
15 aforesaid witness and by the aforesaid
16 attorneys, on the 2nd day of November, 1992.
17 The said witness, being of sound
18 mind and being by me first carefully examined
19 and duly cautioned and sworn to testify the
20 truth, the whole truth, and nothing but the
21 truth in the case aforesaid, thereupon
22 testified as is shown in the foregoing
23 transcript, said testimony being by me
24 reported in shorthand and caused to be
25 transcribed into typewriting, and that the

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1 foregoing pages correctly set forth the
2 testimony of the aforementioned witness,
3 together with the questions propounded by
4 counsel and remarks and objections thereto,
5 and is in all respects a full, true, correct
6 and complete transcript of the questions
7 propounded to and the answers given by said
8 witness; that signature of the deponent was
9 not waived by agreement of counsel.

10 I further certify that I am not of
11 counsel or attorney for either of the parties
12 to said suit, not related to nor interested
13 in any of the parties or their attorneys.

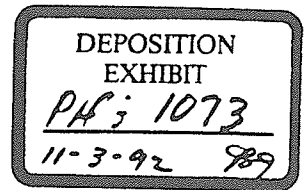
14 Witness my hand and notarial seal
15 at St. Louis, Missouri, this 12th day of
16 November, 1992.

17 My commission expires July 20,
18 1994.

19
20 

21 J. Bryan Jordan

22 Notary Public in and for the
23 State of Missouri



LISTING OF
TOXICITY AND RELATED STUDIES
OF
POLYCHLORINATED BIPHENYLS

NOTE: Those reports listed in addition to those included in Exhibit "C" of the Answers to the Holly Farms Interrogatories indicated by an asterisk.

TRAN 013693

STLCOPCB4029346

()

REPORTS & STUDIES

HARVARD SCHOOL OF PUBLIC HEALTH

1. Experiments to determine the possible toxicity of the following substances:
--chlorinated diphenyl #1268
mixture of chlorinated diphenyl and
chlorinated diphenyl benzene #5460 Dated September 15, 1938

THE BARNARD FREE SKIN AND CANCER HOSPITAL
St. Louis, Missouri

1. Project W-31 Aroclor (1254) Report on Patch Testing,
dated December 22, 1949.

THE KETTERING LABORATORY
Cincinnati, Ohio

1. The Toxicity of the Fogs Formed by Dropping Pydraul F-9, Aroclor 1248, and Tricresyl Phosphate, Upon the Surface of of a Heated Inconel - dated March 11, 1953.
2. The Toxicity of the Vapor of Aroclor 1242 and of Aroclor 1254, dated June 28, 1955.

SCIENTIFIC ASSOCIATES
St. Louis, Missouri

1. The Acute Oral Toxicity (LD₅₀) of Aroclor 1254 for Rats, dated November 10, 1953.
2. The Acute Oral Toxicity (LD₅₀) of Aroclor 1242 for Rats, dated December 11, 1953.

INDUSTRIAL BIO-TEST LABORATORIES, INC.
Northbrook, Illinois

- * 1. Subacute Dermal Toxicity of Aroclor 1221, March 28, 1963.
- * 2. Acute Toxicity Studies With Aroclor 122, Aroclor 5442, and MCS 1016, March 25, 1971. IBT No. A9378.
- 3. Toxicity, Mutagenic, Teratogenic, Reproduction, and Residue Studies As To Various Aroclors in Rats, Chickens, Mice and Dogs - November 17, 1971.
- * 4. Ninety-Day Subacute Oral Toxicity Study With Aroclor 1221 in Beagle Dogs, December 30, 1971. IBT No. C9885.

TRAN 013694

STLCOPCB4029347

Industrial Bio-Test Laboratories, Inc. (continued)

- * 5. Four-Day Static Fish Toxicity Studies With Aroclor 1221, Aroclor 5432, Aroclor 5442, Aroclor 5460, and MCS 1016 In Bluegills and Channel Catfish, January 12, 1972. IBT No. A9380.
- * 6. 90-Day Subacute Oral Toxicity Study With Aroclor 1221 In Albino Rats, April 26, 1972. IBT No. B9888.
- * 7. Toxicity, Reproduction And Residue Study With Aroclor 1221 In White Leghorn Chickens, June 20, 1972. IET No. J900.

BIOLOGICAL CONSULTANTS

Royal D. Suttikus, Ph.D.
Gerald E. Gunning, Ph.D.
New Orleans, La.

- * 1. Interim Report - Fish Residue Analyses dated August 15, 1971.
- * 2. Report covering December 1970, March 1971, June 1971 and September, 1971 - Fish Residue Data - dated June 9, 1972.
- * 3. Final Progress Report, Residue Data, November 1970 to November, 1972 via letter dated February 27, 1973.

YOUNGER LABORATORIES

St. Louis, Missouri

- 1. Toxicological Investigation of OS-95 dated February 22, 1958.
- 2. Toxicological Investigation of OS-95 dated October 20, 1958.
- 3. The Toxicity of the Thermal Decomposition Products of OS-95, dated December 8, 1958.
- 4. Oral Toxicity (LD₅₀) Rats and Skin Absorption (MLD) Rabbits - Various Aroclors, dated June 25, 1962.
- * 5. Toxicological Investigation Of: Aroclor 1221 dated June 13, 1962. Monsanto Project No. Y-62-41. Oral LD₅₀ (rats) and Skin Absorption MLD (Rabbits).
- * 6. Toxicological Investigation of: MCS 1109-Lot KA 801 dated October 27, 1971. Monsanto Project No. Y-71-153. Oral LD₅₀ (Rats, Mixed Sex) and Acute Skin Absorption Minimal Lethal Dose (Rabbits, Mixed Sex); Skin Irritation (Rabbits, Mixed Sex); Eye Irritation (Rabbits, Mixed Sex); Vapor Inhalation (Male Rats).

MONSANTO INDUSTRIAL CHEMICALS COMPANY
Applied Sciences Section
St. Louis, Missouri

- * 1. Determination of Polychlorinated Biphenyl Residues in Albino Rats from a Two-Year Chronic Oral Toxicity Study dated October, 1971 - W. M. Mees, E. S. Tucker, W. J. Litschgi. Special Study 71-7. Job #1348006.
- * 2. Determination of Polychlorinated Biphenyl Residues in Beagle Dog Tissues from a Two-Year Oral Chronic Toxicity Study, dated December, 1972 - W. M. Mees, E. S. Tucker, W. J. Litschgi, J. Cowell. Special Study 71-9. Job #13840.
- * 3. Determination of Polychlorinated Biphenyl Residues in White Leghorn Chickens from a Toxicity, Reproduction and Residue Study with Aroclor 1242, Aroclor 1254, and Aroclor 1260, dated March, 1973 - W. M. Mees, E. S. Tucker, W. J. Litschgi, J. Cowell. Special Study 71-3. Job #1348006.

Monsanto

Medical Department A2SA

DATE October 13, 1971

SUBJECT AROCLOR[®] 1260

cc R. E. Kelly
M. N. Johnson
E. P. Wheeler

REFERENCE

TO : FILE

I spoke with: Renate D. Kimbrough, M.D.
Pathologist
EPA Atlanta Tox. Branch
4770 Buford Highway
Chamblee, Georgia 30341
Tel: (404) 633-3311, ext. 5218

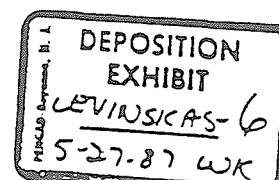
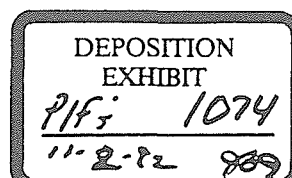
today regarding her observation of bladder tumors in rats fed AROCLOR 1260.

The observations of Vos and Koeman (Toxicol. and Applied Pharmacol. 17, 656-668, 1970) on polychlorinated biphenyls led them to undertake rat reproduction studies with AROCLOR 1254 (Lot AK38) and 1260 (Lot AK3). The materials were obtained from Glasgow at FDA. Their primary findings have been lesions in the liver, and Dr. Kimbrough plans to present a paper on the liver lesions at the 1972 spring meeting of the Society of Toxicology. In addition, she has seen 2 lesions in the bladders of rats fed AROCLOR 1260. The first occurred in a female which died after 6 months on test. This was diagnosed as a malignant anaplastic carcinoma of the bladder. The second was in a male killed after 8 months on test. The first diagnosis was of epithelial hyperplasia. Sections of both bladders were sent to NCI for diagnosis to Drs. Strauss (?) and Katherine Snell. The diagnosis of carcinoma in the female was confirmed. The lesion in the male was not resolved. Some pathologists believe it is a carcinoma, others believe it is hyperplasia.

Dr. Kimbrough stated that they were trying to analyze the AROCLOR 1260 sample for impurities, but were having some difficulty with their equipment. I indicated that we would try to track down material from the sample that they had received. In the event that we cannot locate this lot, we probably can get some material back through Thomas B. Gaines, Supervisory Research Pharmacologist, at the same location as Dr. Kimbrough. If we have an analysis of this lot, we should make the results known to Dr. Kimbrough.

Dr. Kimbrough said that they had observed porphyria in some rats with both compounds. She also indicated that they were contemplating doing a 2 year feeding study with larger numbers of rats to investigate the problem of bladder tumors. She is perplexed by the bladder tumors, and she is not emphasizing them in her discussions. However, she has mentioned her findings when PCB's have been discussed at various interagency meetings. This is apparently how Weissburg learned about them. (cont'd.)

10-10 REV 11-68



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FILE
October 13, 1971
Page - 2 -

As a final note, Dr. Kimbrough expressed concern over whether PCB's presented an additional hazard to the employees who manufacture it. I told here that because of the chloracne and liver hazards, there had been medical supervision of the employees. However, I would raise this point with Drs. Kelly and Johnson for their review.

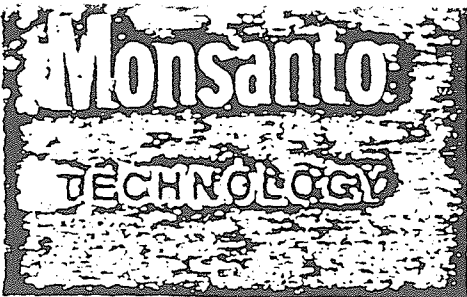

George J. Levinskas

/bks

SCM 052788

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STLCOPCB4029351



DMEH
Toxicology Section/St. Louis
(CO./DIV./DEPT./LOCATION)

SPECIAL REPORT
(TYPE OF REPORT)

REPORT NO.: MSL-2002

JOB/PROJECT NO.:

DATE: October 14, 1981

TITLE: TOXICITY OF AROCLOR PRODUCTS 1242, 1254 AND
1260 TO THE LIVER OF ALBINO RATS

AUTHORS: George J. Levinskas, Ph.D.

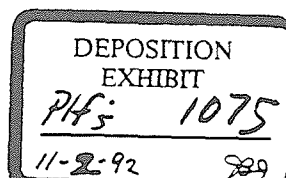
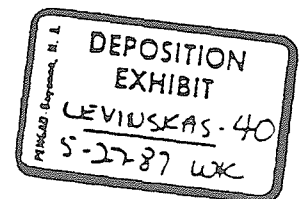
ABSTRACT: This report is a tabulation of the results
of microscopic evaluation of liver sections
from rats fed AROCLOR 1242, AROCLOR 1254 or
AROCLOR 1260 for two years.

AUTHORS: George J. Levinskas, Ph.D.
TITLE: TOXICITY OF AROCLOR PRODUCTS 1242, 1254
AND 1260 TO THE LIVER OF ALBINO RATS

REPT. NO.: MSL-2002

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- 10 - J.M. Norris, Dow Chemical
- 11 - R.A. Stohr, B3NJ

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INTRODUCTION

The polychlorinated biphenyls (PCBs) comprise a family of compounds of variable chlorine content. PCBs manufactured by Monsanto (tradenamed "Aroclor") bear a four digit number, the '12' indicating biphenyl and the last two digits indicating the chlorine content by weight percent. Since the chlorine atoms are randomly distributed among the 10 ring positions available for substitution, each material is a mixture of isomers.

In 1969, Monsanto sponsored a series of animal studies at Industrial BIO-TEST Laboratories in Northbrook, Illinois, on Aroclor 1242, 1254, and 1260 for the assessment of the health and environmental hazards of these materials. This series consisted of 2-year chronic feeding studies to rats and dogs, a 3-generation rat reproduction study, a rat teratology study, a dominant lethal mutagenic study in mice and a toxicity/reproduction study in chickens on each of the 3 Aroclor products. Even though no such action was contemplated, such a broad battery of tests would have been adequate to support Food Additive Petitions for each of these materials. These studies were initiated by reports that PCBs had been detected in the environment. A report (Nelson, 1972b) by a Panel on Hazardous Trace Substances of an Ad Hoc Committee on Environmental Health Research covers the development of information on the environmental and biological effects of PCBs. There have been subsequent literature reviews which need not be recapitulated here [DHEW, 1978; EPA, 1976; IARC, 1978; NAS, 1979; Roberts, et al. (1978)].

Starting in 1969, while these studies still were in progress, information regarding them was made available to various government groups.¹ Subsequently, data from these studies were presented at a conference

sponsored by the National Institute of Environmental Health Sciences at Rougemont, N.C. on December 20-21, 1971, but the account of these studies was omitted from the published proceedings (Keplinger, et al., 1972; Nelson, 1972a).

Subsequently, it was reported that female Sherman strain rats developed hepatocellular carcinomas when fed Aroclor 1260² from Lot No. AK-3; the same lot used for the earlier Monsanto study with this material. As a result, Monsanto requested the contract laboratory's pathologists to review livers from all available rats from the 3 Aroclor studies. That review (which could have included new sections of liver from animals examined previously in addition to sections from animals not examined previously) was presented in the reports of Gordon and Richter (1975a,b,c).

In November 1975, reports containing evaluations of liver sections from the 2-year rat feeding studies (Gordon and Richter, 1975a,b,c) and the results of an independent evaluation of the same liver sections (Pour, 1975) were presented to and discussed with several federal regulatory agencies³. Later that month, a summary of data from all of the studies was presented at the National Conference on Polychlorinated Biphenyls sponsored by the Environmental Protection Agency and held in Chicago on November 19-21, 1975 (Calandra, 1976). Only summaries of data from these and other toxicity studies conducted over the years have been published (Jenkins, et al., 1972; Keplinger, et al., 1971; Monsanto, 1980). Knowledge of these efforts may have prompted the statement in a recent article that "...Monsanto, whose reaction to the possibility that polychlorinated biphenyls (PCBs) might be an ecological disaster was a classic of how such issues should be handled, says Ford⁴. Monsanto began to investigate the dangers as soon as they were

seriously voiced. It insisted on keeping an open mind about them. As soon as evidence appeared that there was a strong chance PCBs were a hazard, it published the results of its investigations, admitting the danger. It thus established a reputation of honesty even among the environmentalists." (Clutterbuck 1981).

At a later meeting in Bethesda, MD.⁵, pathologists selected and reviewed some liver slides from the Monsanto-sponsored and Kimbrough studies. The pathologists differed in the terminology used to classify the lesions. They did conclude that the general incidence and severity of liver lesions (hyperplasia, nodular hyperplasia and neoplastic changes - carcinomas) were greater in the rats from the study subsequently published by Kimbrough, et al. (1975). No carcinomas were seen in the Monsanto-sponsored studies. It was noted that either the strain of rat or sex could have accounted for the differences. The spontaneous incidence of hyperplastic or neoplastic liver lesions in the Sherman strain rat was unknown, and the occurrence of such lesions appears to be higher in female rats and mice.

The different diagnoses were discussed with Dr. Philippe Shubik of the Eppley Institute for Research in Cancer at the University of Nebraska. Dr. Shubik suggested that the liver sections from these studies could be reviewed by Dr. Parvis Pour. This was done.

This publication is an effort to make readily available information in the Gordon and Richter reports (1975a,b,c) which are only summarized in the literature (Calandra, 1976).

METHODS

It appears that the Threshold Limit Value of 0.5 mg/m^3 for chlorobiphenyl with 54% chlorine (ACGIH, 1969) was used to estimate suitable dose levels for the rat feeding studies. For that purpose the following assumptions were made: if the ambient air concentration of PCBs is 0.5 mg/m^3 , and if all of the inhaled PCBs are absorbed, and if 15 m^3 of air are inhaled during the working day, a person would receive a PCB dose of 7.5 mg/day . The corresponding dosage would be approximately 0.1 mg/kg/day for a 70-kilogram person. Consequently, dosages of 0.1, 1, and 10 mg/kg/day were decided upon, and the dietary concentrations were set at 1, 10, and 100 ppm. As it turned out, the assumptions used to set dosages for the feeding study resulted in the low dose level rats receiving a dosage that was 100 times higher than the 0.001 mg/kg/day which FDA later calculated as allowable for protracted ingestion based on human data (FDA, 1973).

For the chronic toxicity study in rats⁶, weanling animals of the Charles River CD strain⁷ were divided into a control group and 9 treatment groups, each consisting of 50 males and 50 females, with 3 treatment groups assigned to each of the 3 Aroclor products studied (1242, 1254, and 1260). Not all of these animals started at the same time. After about 2 months on test, additional groups of males and females were assigned to each test diet and the controls. These animals were added to allow a sufficient number of animals for sacrifices at 3, 6 and 12 months to provide some information prior to the completion of the 2-year study period. ~~The numbering sequence~~

~~and the designation of some animals as "Extra"~~ suggests that additional animals were placed on test.

Groups of 5 males and 5 females were scheduled to be sacrificed after 3, 6, and 12 months of feeding, and survivors were to be sacrificed at the end of the test period.⁸ Animals were to be given a gross autopsy and tissues were to be preserved for possible histologic examination. Tumors or lesions suggestive of tumors were to be examined.

RESULTS

Data from the Gordon and Richter reports (1975a,b,c) on liver slides are shown in Tables 1, 2, and 3 for Aroclor 1242, 1254, and 1260, respectively.⁹ While the text of the reports contained comments specific to the particular Aroclor, they also contained similar comments such as "There is evidence of a chemical effect on the liver.....This consists of a hepatocellular alteration beginning as focal hypertrophy which progresses to nodular hyperplasia, and in a few animals to hepatoma or cholangiohepatoma" and "In the absence of metastasis, invasiveness, severe basophilia, mitoses, or other evidence of anaplasia; these are benign tumors rather than malignant carcinomas. There was no evidence of metastasis or invasiveness of these tumors in this study". The reports also stated that "The other treatment-related lesions reported are regarded as degenerative or hyperplastic in nature and they are morphologic manifestations of an adaptive response of the liver associated with biotransformation of the test material" and "In most instances, the spectrum of treatment-related histopathological findings in the liver from this re-evaluation did not differ significantly from that previously reported in our original report.....No hepatocellular carcinomas were observed".

With respect to Aroclor 1254, it was noted that "One liver tumor (a hepatoma) previously reported in a T-II animal (No. 445) was re-classified as nodular hyperplasia" (Gordon and Richter, 1975a)¹⁰.

DISCUSSION

The initial reports of these studies concluded that the target organ was the liver for each Aroclor product. This was confirmed by the second evaluation of liver slides (Gordon and Richter, 1975a,b,c). These alterations were slow to develop, their incidence being related to both dose level and duration of treatment. Since there were increased incidences of vacuolar changes in the cytoplasm of the hepatocytes and focal hypertrophy at all dose levels for all 3 Aroclor products at the 24-month sacrifice, a "no-effect" level was not established for liver effects.

With respect to the slides from Industrial BIO-TEST, Pour (1975) concluded that Aroclor 1242, 1254, and 1260 "showed a dose-dependent hepatotoxic effect, characterized by degenerative and regenerative processes. With one exception, all lesions were considered non-neoplastic. Structures similar to cholangiocarcinomas and hepatomas were found in one rat. However, the possibility of metastases of a carcinoma into the liver has been also considered." In the report, he noted one lesion which seemed to "represent a metastatic tumor (probably a mammary gland carcinoma of the same animal from which ...[the particular liver section]... was submitted), rather than a cholangiocarcinoma". This occurred in an animal fed 100 ppm of Aroclor 1254. The contract laboratory pathologists diagnosed the presence of mammary tumor metastases in the liver of this rat (Gordon and Richter, 1975b).¹¹

SUMMARY and CONCLUSIONS

Groups of male and female rats were fed diets containing either 1, 10, or 100 ppm of one of 3 Aroclor products, 1242, 1254, or 1260, for 2 years. Focal hypertrophy of the liver occurred at 3, 3, and 12 months for rats fed Aroclor 1260, 1254, and 1242, respectively. Focal hypertrophy was not seen in livers of control animals. At the 24-month sacrifice, livers of animals from all dose levels of the 3 Aroclor products had an increased incidence of vacuolar changes and focal hypertrophy. Livers of some animals fed 100 ppm of each Aroclor also had benign liver tumors (hepatoma or cholangiohepatoma). No hepatocellular carcinomas were observed.

Footnotes

1. Dates of initial correspondence and contacts.
October 3, 1969. E.P. Wheeler to A.R. Glasgow, Division of Pesticides. Food and Drug Administration.
April 8, 1970. R.E. Kelly to H. Blumenthal, Petitions Review Branch, Food and Drug Administration.
April 22, 1970. E.P. Wheeler to E.J. Burger, Jr., Office of Science and Technology.
June 1, 1970. E.P. Wheeler to H.E. Stokinger, Bureau of Occupational Safety and Health.
2. R.D. Kimbrough, personal communication.
3. November 13-14, 1975. Council on Environmental Quality.
Environmental Protection Agency.
Food and Drug Administration.
House Subcommittee Manpower, Compensation, Health and Safety.
National Cancer Institute.
National Institute for Environmental Health Sciences.
National Institute for Occupational Safety and Health.
4. Dr. David Ford of Bath University.
5. At National Cancer Institute on January 21, 1975. Present were D.E. Gordon, R.D. Kimbrough, G.J. Levinskas, R.A. Squire, W.R. Richter.
6. Since the events described earlier, the validity of many toxicity studies conducted by Industrial BIO-TEST Laboratories has been challenged. Therefore, the available raw data supplied by Industrial BIO-TEST was reviewed to determine whether this study could be validated. The review showed that the data bases (including the lack of a protocol) were insufficient for a complete validation of the study. It was decided to focus on presenting the primary liver effects reported by Gordon and Richter (1975a,b,c). Therefore, a complete audit was not undertaken. Available records were examined for a determination that the animals were placed on test and of their ultimate fate. Necropsy and microscopic reports were also examined for findings pertaining to livers of those animals. Significant discrepancies which were found between data in Tables 1, 2, and 3 and the data base for the Gordon and Richter reports are noted in this report. On some records, the labels Aroclor 1242 and Aroclor 1260 are interchanged as determined by a check of the animal numbers.
7. Charles River Breeding Laboratories, North Wilmington, Massachusetts.
8. Animals sacrificed at 6 and 12 months were placed on test about 2 months after the first group. Those sacrifice periods are sometimes labeled 8 and 14 months, respectively, which corresponds to the calendar time from the start of the test but overstates the time on test for those groups.

9. As a result of the examination described in footnote 6, a few animals were reassigned to other dose levels on basis of assigned numbers. Three with no recorded liver lesions were deleted from the 24-month listing because of short duration on test: 14 months at 100 ppm Aroclor 1242, 15 months at 10 ppm Aroclor 1254 and 13 months at 100 ppm Aroclor 1254. Therefore, numbers in Tables vary slightly from those in reports of Gordon and Richter (1975a,b,c).
10. That change and comments in the footnotes to the Tables were noted during the examination described in footnote 6. In addition, the following also were noted during the examination.

Aroclor 1254 - 100 ppm animal marked "Extra³" with diagnosis of hepatoma in record of examination for original Industrial BIO-TEST report. Animal not listed in Gordon and Richter report.

- 100 ppm animal no. 542 with gross autopsy notation that liver appears tumorous and white foci has no record of examination.

Aroclor 1260 - 100 ppm animal no. 293 with gross autopsy notation of tumor on liver has no record of examination.

In some instances, diagnoses were crossed out on the available records. These were included in the Tables. Crossed out diagnoses for hepatoma or cholangiohepatoma are noted in the footnotes to the Tables.

11. Dr. Pour's report does not include the following liver sections noted by discrepancies between his tabulation and the Gordon and Richter reports.
- 1 from control at 12 month sacrifice,
 - 5 from 100 ppm Aroclor 1242 at 24 months,
 - 1 from 100 ppm Aroclor 1260 at 3 months.

None of these animals were reported as having hepatomas or cholangiohepatomas in the Gordon and Richter reports.

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1. ACGIH (1969). Threshold Limit Values of Airborne Contaminants. American Conference of Governmental Industrial Hygienists. Cincinnati.
2. Calandra, J.C. (1976). Summary of toxicological studies on commercial PCB's. In: National Conference on Polychlorinated Biphenyls, November 19-21, 1975. Chicago, Illinois. EPA Report No. 560/6-75-004. March. NTIS PB-253 248. pp.35-42.
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Table 1

Aroclor 1242: Primary Liver Lesions Among Albino Rats

Sacrifice Interval	3 Months				6 Months				12 Months				Terminal ^a			
Diet Level (ppm)	0	1	10	100	0	1	10	100	0	1	10	100	0	1	10	100
No. Animals Examined	10	8	10	9	10	10	8	9	10	10	10	9	23	32	29	19
<u>Findings</u>																
Vacuolar change	2	0	2	1	1	0	0	0	1	2	4	0	1	7	8	9
Focal necrosis	0	1	0	0	1	1	0	0	0	0	0	0	1	1	1	0
Focal lymphoid infiltration	0	0	0	0	0	3	0	0	0	0	1	0	1	0	0	0
Focal hypertrophy hepatocytes	0	0	0	0	0	0	0	0	0	0	0	1	0	2	3	8
Nodular hyperplasia	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	8
Ductular hyperplasia	0	0	0	1	0	0	0	0	1	0	1	0	5	3	3	3
Hepatoma	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	3 ^b
Cholangiohepatoma	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1 ^c

^a Control group has animal dead at 19 months and 2 marked "Extra".

1 ppm group has animals dead at 19, 22, 22, 23, 23 months.

10 ppm group has animals dead at 23, 23 months.

100 ppm group has animals dead at 22, 22, 22 months and 2 marked "Extra".

^b Includes 1 animal without record of examination in original IBT report. Not listed as hepatoma in worksheets for Gordon and Richter report but appears and was crossed out on typed tabulation for animal marked "Extra". Diagnoses for other 2 animals do not appear in records of examinations for original IBT report.

Diagnosis does not appear in records of examinations for original IBT report.

Table 2

Aroclor 1254: Primary Liver Lesions Among Albino Rats

Sacrifice Interval	3 Months				6 Months				12 Months				Terminal ^a			
Diet Level (ppm)	0	1	10	100	0	1	10	100	0	1	10	100	0	1	10	100
No. Animals Examined	10	10	10	10	10	10	10	10	10	10	10	10	23	30	26	26
<u>Findings</u>																
Vacuolar change	2	3	3	1	1	0	0	0	1	1	4	1	1	7	9	13
Focal necrosis	0	1	1	0	1	2	0	2	0	0	0	1	1	3	1	1
Focal lymphoid infiltration	0	0	0	1	0	0	0	2	0	0	2	1	1	2	0	1
Focal hypertrophy hepatocytes	0	0	0	1	0	0	0	4	0	0	4	2	0	3	4	14
Nodular hyperplasia	0	0	0	0	0	0	0	0	0	0	0	0	1	0	3	14
Ductular hyperplasia	0	0	0	0	0	0	0	0	1	1	1	0	5	6	3	4
Hepatoma	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4 ^b
Cholangiohepatoma	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2 ^c

^a Control group has animal dead at 19 months and 2 marked "Extra".

1 ppm group has animals dead at 22, 22 months, 1 marked "Extra" and 1 other for which date of death is not recorded.

10 ppm group has animals dead at 22, 22, 22 months.

100 ppm group has animals dead at 23, 23 months and 9 marked "Extra".

^b Includes 2 animals marked "Extra". Diagnoses do not appear in records of examination for original IBT reports.

^c Both animals marked "Extra" with same designation. 1 without apparent record of examination for original IBT report. Diagnosis for other animal does not appear in records of examinations for original IBT report.

Table 3
Aroclor 1260: Primary Liver Lesions Among Albino Rats

Sacrifice Interval	3 Months				6 Months				12 Months				Terminal ^a			
Diet Level (ppm)	0	1	10	100	0	1	10	100	0	1	10	100	0	1	10	100
No. Animals Examined	10	10	10 ^b	10	10	6	5	9	10	9	10	9	23	26	25	25
<u>Findings</u>																
Vacuolar change	2	0	2	3	1	1	2	6	1	2	5	3	1	5	7	9
Focal necrosis	0	0	4 ^c	0	1	0	1 ^d	0	0	0	0	0	1	4	3	6
Focal lymphoid infiltration	0	0	0	0	0	2	2	0	0	0	0	0	1	0	1	0
Focal hypertrophy hepatocytes	0	0	0	2	0	0	0	3	0	0	0	4	0	3	13	9
Nodular hyperplasia	0	0	0	0	0	0	0	0	0	0	0	1	1	0	7	6
Ductular hyperplasia	0	0	1	0	0	0	0	0	1	1	0	2	5	6	7	12
Hepatoma	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1 ^e	7 ^{f,g}
Cholangiohepatoma	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4 ^{g,h}

^a Control group has animal dead at 19 months and 2 marked "Extra".

1 ppm group has animals dead at 20, 22, 22 months.

10 ppm group has animals dead at 19, 22, 22 months and 1 marked "Extra".

100 ppm group has animals dead at 22, 22 months.

^b Includes 1 marked "Extra".

^c Worksheets show listings under this diagnosis with arrow pointing to Vacuolar change column.

^d Date of death of this animal not recorded.

^e No record of examination for original IBT report. Listed on worksheets for Gordon and Richter report with diagnosis crossed out.

^f Includes 2 animals without record of examination for original IBT report. Diagnoses for other 5 animals do not appear in records of examinations for original IBT report. 2 of these diagnoses crossed out on worksheets for Gordon and Richter reports.

^g Includes one animal with both diagnoses. Hepatoma is 1 of 2 crossed out (superscript f).

^h Includes 3 animals without record of examination for original IBT report. Diagnosis for other animal does not appear in records of examinations for original IBT report. 2 of these diagnoses crossed out on worksheets for Gordon and Richter reports.

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for EPA
1/9/76
File PCB ECH

Toxicity and Environmental Effects of Commercial PCBs

Introduction

Polychlorinated biphenyls (PCBs) are not a single chemical entity. They are produced by chlorinating biphenyl to achieve a final product with specified properties. These products, sold under the trade name AROCLOR by Monsanto, are mixtures of chlorine-containing biphenyls with different numbers and positions of the chlorine substituents.

Over the years, a series of investigations have been conducted to assess potential health and environmental hazards of PCBs. The 3 predominant commercial mixtures (AROCLORS 1242, 1254 and 1260) have been studied most intensively. Toxicity tests performed on these 3 commercial mixtures were typical in scope of those designed for the evaluation and establishment of the safety of direct and indirect food additives.

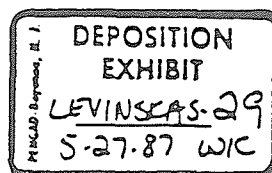
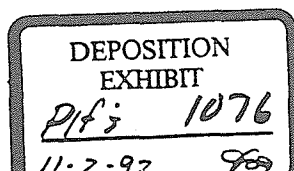
It may be noted that AROCLOR 1260 no longer is produced in this country since it does not meet the physical specifications for its restricted uses.

Conclusions Based on Monsanto Studies

1) As a class, the AROCLORS are relatively harmless materials for routine industrial handling under ambient conditions.

2) Threshold Limit Values of 1 mg/m^3 and 0.5 mg/m^3 have been established for materials containing average chlorine values of 42% and 54%, respectively, of the available sites.

3) The no-effect level for these materials in chronic



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rat and dog feeding studies is about 10 ppm in the diet.
No hepatocellular carcinomas were present.

4) The no-effect level on rat reproduction is between 1 and 10 ppm in the diet since higher levels result in low mating indices.

5) No teratogenic or mutagenic effects were observed in studies with rats and mice.

6) In chicken reproduction and teratology studies, AROCLOR 1242, which produced the most severe effects, had a no-effect level of 2-4 ppm in the diet.

7) Acute toxicity to fish varies from less than 1 ppm to greater than 100 ppm depending on the specific AROCLOR and species of fish tested.

8) PCBs containing 3 or less chlorine substituents per molecule are reasonably biodegradable.

Summary of Monsanto's Long-term Toxicity Studies on Commercial PCBs.

These tests consisted of 2-year (lifetime) feeding to rats, 2-year feeding to dogs, 3-generation rat reproduction studies with 2 litters cast per generation, rat teratology studies and dominant lethal mutagenic studies in mice. Toxicity and reproduction studies in chickens were performed to evaluate possible untoward effects in birds such as decreases in eggshell thickness. In addition, biodegradation and tissue accumulation studies have been conducted.

The highest dietary level (100 ppm) in the lifetime rat feeding studies produced a weight depression at 24 months in females fed AROCLOR 1254 and liver weight increases in all

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groups except males fed AROCLOR 1242. As with other halogenated hydrocarbons, the important histopathologic changes were present in the livers of animals sacrificed at the end of the study. These consisted of hepatocellular alterations such as focal hypertrophy, cytoplasmic lipid changes and in some animals from the 100 ppm groups, hepatomas or cholangiohepatomas. No evidence of hepatocellular carcinogenicity of the AROCLORS was found.

In the dog 2-year studies, some slight decreases in body weight gain were noted. At the highest feeding level (100 ppm), AROCLOR 1260 produced an increase in serum alkaline phosphatase activity and liver weights without concomitant histopathologic changes. However, there was evidence of gastrointestinal inflammatory lesions and ulcerations which appear to be similar to those found by Allen in rhesus monkeys fed AROCLOR 1248. (AROCLOR 1248 was an experimental product which never was commercialized.)

In the rat reproduction studies, none of the AROCLORS produced adverse effects in the 2 litters of the first generation. In the second and third generations, there was a reduction in the mating index at the 2 highest feeding levels (10 ppm and 100 ppm). The ability of females to conceive, carry the delivery process to parturition, and to successfully nourish the young was not affected by the 3 AROCLORS. No changes were produced in the reproductive tracts of either male or female rats by any of the 3 AROCLORS.

There was no evidence of teratogenic or mutagenic changes with any of the PCBs at maximally tolerated dose levels in

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rats and mice.

In the chicken toxicity/reproduction study, AROCLOR 1260 was without effect at all test levels. AROCLORS 1242 and 1254 at 100 ppm in the diet decreased egg hatchability. In fact, poor hatchability of eggs was found from hens fed 8 ppm of AROCLOR 1242. In addition, AROCLOR 1242 at 10 and 100 ppm and AROCLOR 1254 at 100 ppm were associated with reduced eggshell thickness. Chick viability was affected by both substances at a dietary level of 10 ppm.

Biodegradation studies were conducted using semi-continuous activated sludge systems and tissues of rats fed PCBs were analyzed to measure accumulation and retention of these materials. These factors are influenced by the number and position of the chlorine substituents. Higher chlorine-containing members are more resistant to biodegradation and they accumulate more readily and are less readily metabolized and/or excreted from lipid tissue in animals. PCBs containing 3 or less chlorine substituents per molecule are reasonably biodegradable.

Comments

The conclusion that "No evidence of hepatocellular carcinogenicity of the AROCLORS was found" in the 2-year rat feeding study was reached only after extensive re-evaluation of the original liver slides as well as additional liver sections from all of the animals after the Kimbrough results on AROCLOR 1260 became known to us. The slides were read

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independently by Dr. D. Gordon of Industrial BIO-TEST Laboratories, Professor W. Richter of the University of Chicago, and by Professor P. Pour of the Eppley Institute for Research in Cancer. In addition, Dr. Pour evaluated the Kimbrough slides and does not agree with the reported findings.

Barsotti and Allen have reported that 2.5 ppm of AROCLOR 1248 in the diet of rhesus monkeys adversely affected reproduction. This approximates a dosage of 100 $\mu\text{g/kg/day}$. It is higher than the safe human intake (1 $\mu\text{g/kg/day}$) estimated by FDA from human data when it promulgated its tolerances for PCBs in food.

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Human data

(F.R. July 6, 1973. p. 18097, Section (2))

Lowest level of PCBs producing effect in man = 500 mg total.
500 mg consumed by 50 kg individual over 50 days.

$500 \text{ mg} \div 50 \text{ kg} \div 50 \text{ days} = 200 \text{ } \mu\text{g/kg/day}$ (effect level)

Using 10-fold safety factor leads to estimate that 20 $\mu\text{g/kg/day}$ may be safe over a 50-day interval.

Since PCBs have long half-life; calculating same intake over a 22-month span arrives at an estimated safe intake for a protracted period of 1 $\mu\text{g/kg/day}$.

Primate data

(Fed. Proc. 34:338 (1975). Abstract 675. Barsotti & Allen)

At 2.5 ppm in diet, primates ingested 182 mg over 52 weeks.
 $182 \text{ mg} \div 365 \text{ days} = 0.5 \text{ mg/day}$ which produced effects.

Assuming 5 kg primate, $500 \text{ } \mu\text{g} \div 5 \text{ kg} = 100 \text{ } \mu\text{g/kg/day}$

Conclusions: Primate study done at dosages (100 $\mu\text{g/kg/day}$) greater than the safe dosage (1 $\mu\text{g/kg/day}$) estimated from human data.

Primate study does show effects at lower dosages over longer time span (100 $\mu\text{g/kg}/365 \text{ days}$) than had been observed in man (200 $\mu\text{g/kg}/50 \text{ days}$).

The total amount of PCBs producing effects in primates (182 mg) was lower than that producing effects in man (500 mg).

(N.B. Abrahamson & Allen, Env. Health Perspectives. June, 1973, 81-86. Report that infant monkeys are able to tolerate doses of PCBs that produce extreme morbidity in adult monkeys.)

Dog and Rodent Data

(F.R. July 6, 1973. p. 18097, Section (1))

"No-effect" level for man using a 100-fold safety factor and accepting 10 ppm as a "no-effect" level in animals would be 2.5 $\mu\text{g/kg/day}$ based on dog data and 3 $\mu\text{g/kg/day}$ based on rat data.

If rat data "no-effect" level drops to 1 ppm, then corresponding estimate of human "no-effect" level drops to 0.3 $\mu\text{g/kg/day}$.

Question: In a "collision" between rat data using a 100-fold safety factor and human data using a 10-fold safety factor, which data base would you like to be riding on?

Comment: Since human data is available, it could be argued that the traditional 100-fold safety factor is not necessary. Application of a 30-fold safety factor to rat data, supported by dog and human data, makes present estimate of safe human intake appear reasonable.

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PCB Primate and Human Residue Data

	<u>Primates</u>		<u>Human</u>
Conc. in diet, ppm	25	2.5	-
Intake, ug/kg/day	800	100	-
Estimated safe dose, ug/kg/day		-	-/
Liver conc., ug/g	56.3(0.01)*		-
Fat conc., ug/g	50.0(27.7)*		0 (314 samples)**
			<1 (125 samples)
			1-2 (165 samples)
			>2 (33 samples)

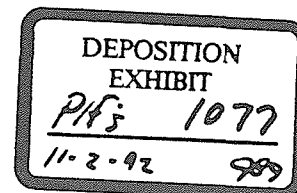
*Value in () from infant primate.
Allen et al., TAP 30: 440-451 (1974)

**Yobs, Env. Health Persp., 79-81, April 1972

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PROGRESS REPORT
ORGANIC CHEMICALS DIVISION
TECHNOLOGY DEPARTMENT



W. M. Mees W. J. Litschgi J. D. Hinchey E. S. Tucker	FOR January 1972	DETAILS NO	REPORT NO 5	DATE 1-31-72
LOCATION St. Louis - South Second Street	JOB NOS 2-21-760.01-13480, 85010, 85050			

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PROJECT TITLE
ENVIRONMENTAL ANALYTICAL PROGRAM - TOXICOLOGY SUPPORT STUDIES

PROJECT OBJECTIVE
Determination of the type and level of polychlorinated biphenyls (PCB's) obtained in the tissues of laboratory animals fed Aroclor 1242, Aroclor 1254, Aroclor 1260, Aroclor 1016, and Aroclor 1221 for correlation with toxicological effects.

SUMMARY
The PCB residue levels found in selected chicken tissues and eggs from the "Toxicity, Reproduction, and Residue Study of Aroclor in White Leghorn Chickens" (IBT J7300) were subjected to a multiple regression analysis. The statistical analysis was used to develop an equation for predicting the PCB residues in the remaining tissues. From the predicted PCB residues the following conclusions were drawn.

The average PCB lipid storage level found in the tissues (male and female), for each product after six months exposure are shown in the following table.

PCB Lipid Storage Level (PPM)

Feed Level	1 PPM	10 PPM	100 PPM
Aroclor 1242	28	196	2356
Aroclor 1254	53	317	4080
Aroclor 1260	65	417	5176

Tissue and sex differences were also apparent in the data; the following patterns were observed:

- 1) At low exposure, muscle>fat>liver.
- 2) At high exposure, muscle>liver>fat.
- 3) All cases, male>female.

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PCB residues in the muscle, fat and egg tissues from animals fed the 30-day recovery diet were significantly lower, while the PCB residues in the liver were only slightly less. Remobilization of the residues in the animals subjected to the highest exposure was noticeably slower.

Muscle and liver tissues have been excised and composited from the 30-day chicks collected during this study. Analysis of these chick tissues has been completed and the PCB residues are being calculated at this time.

Analysis of selected chicken tissues, 30-day chick tissues and eggs from the supplementary study (IBT 8746) with Aroclor 1242 at lower exposures has been completed. The complete clean-up procedure was required with the chicken tissues to remove several interferences to the accurate quantitation of the PCB residues on the EC/GC. The PCB residues are being calculated for comparison with the predicted residues from the equations developed in the study (IBT J7300) above.

Analysis of the tissues from dogs fed Aroclor 1016 and 1221 is complete and the PCB residues are being calculated. The complete clean-up procedure was again required to remove several interferences to the quantitation of the PCB residues, confirming our experience with the two-year dogs. Extensive alterations of the homolog distributions of these less chlorinated products are being observed on the EC/GC chromatograms.

Analysis of the tissues from rats fed Aroclor 1016 is complete and the PCB residues are being calculated. An abbreviated clean-up procedure, an alumina column treatment, was sufficient to remove any interferences to the quantitation of the PCB residues in these rat tissues. Again extensive alterations of the homolog distribution of this product are being observed.

With the rats fed Aroclor 1221 low levels of PCB residue eluting in the Aroclor 1260 region of the chromatogram were observed. At these low levels several interferences become significant and the extracts required the complete clean-up procedure before accurate quantitation of the PCB residues could be carried out. Analysis of the rat tissues is complete and the PCB residues are being calculated.

The EC/GC procedure for PCT's has been extended to biological samples. Using chloroform for the extraction solvent, recoveries of 85% or better were achieved. Selected samples from the 90-Day Subacute Toxicity Studies of Aroclor 5432 and 5442 in rats and dogs have been extracted. EC/GC analysis of the extracts and calculation of the PCT residues should be complete by 3-15-72.

Analysis of the samples from the Subacute Toxicity Studies of Aroclor 1016 and 1242 in catfish and bluegills have been started. These studies, encompassing seven levels of exposure in the range of 0.014 ppm to 0.300 ppm, were carried out at Bionomics, Inc. of Wareham, Massachusetts.

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UTURE PLANS

Analysis of selected tissue samples for the level and type of PCB residues retained from the following feeding studies carried out by Industrial Bio-Test Laboratories.

	<u>Tissues Available</u>
90-Day Subacute Oral Toxicity Study of MCS 1109 with Beagle Dogs	2-15-72
90-Day Subacute Oral Toxicity Study of MCS 1109 with Albino Rats	2-15-72
Toxicity, Reproduction, and Residue Study of Aroclor 1016 with White Leghorn Chickens	7-72
Toxicity, Reproduction, and Residue Study of Aroclor 1221 with White Leghorn Chickens	7-72

W. M. Mees

E. S. Tucker

db
2-2-72

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PROGRESS REPORT
ORGANIC CHEMICALS DIVISION
TECHNOLOGY DEPARTMENT

DEPOSITION
EXHIBIT

PH: 1078

11-2-92

89

ORDERED BY W. M. Mees W. J. Litschgi J. D. Hinchey E. S. Tucker	FOR November- December 1971	DETAILS NO	REPORT NO. 4	DATE 11-30-71
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PROJECT TITLE

ENVIRONMENTAL ANALYTICAL PROGRAM - TOXICOLOGY SUPPORT STUDIES

PROJECT OBJECTIVE

Determination of the type and level of polychlorinated biphenyls (PCB's) retained in the tissues of laboratory animals fed Aroclor 1242, Aroclor 1254, and Aroclor 1260 for correlation with toxicological effects.

SUMMARY

Analysis of selected tissue samples from the "Two Year Chronic Oral Toxicity Study of Aroclor in Beagle Dogs" has been completed and the data subjected to a multiple regression analysis. The predicting equation obtained indicated that, relative to the two rat studies completed earlier, there was more scatter in these data points. This increased variability is understandable in light of the fact that much lower storage levels were observed and that the tissue samples from the recovery sacrifices were from individual animals rather than the group composite samples analyzed in the rat study.

The analysis of several additional selected samples reduced the equation's variability to an acceptable level and the storage levels in the remaining tissues were calculated. Even with the analysis of the several additional samples, a savings of \$3700 was realized.

The following conclusions were drawn from the data. The average PCB lipid storage levels found in the tissues for each product after two years exposure are shown in the following table.

PCB Lipid Storage Level (PPM)

Feed Level	1 PPM	10 PPM	100 PPM
Aroclor 1242	3	5	16
Aroclor 1254	6	18	109
Aroclor 1260	13	86	579

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It should be noted that the above storage levels are at least an order of magnitude less than the storage levels observed with the rats.

The storage levels in the animals placed on the recovery diet decreased to approximately one-half the 24 month storage level in 60 days.

Alterations of the homolog distributions were observed with all of the products fed and were much more extensive than previously noted in any of the other tissue residue studies.

Selected tissue samples from the "Toxicity, Reproduction, and Residue Study of Aroclor in White Leghorn Chickens" (IBT J7300) have also been analyzed and the data subjected to multiple regression analysis. Acceptable equations for predicting tissue residues and egg residues were determined, and the tissue storage levels predicted in the remaining samples. The excising, compositing and analysis of the chick samples collected from this study will be completed shortly. A savings of ~\$8000 in analytical costs was realized using regression analysis and selected data points.

Samples from the "Toxicity, Reproduction and Residue Study of Aroclor 1242, Lot AK-255 in White Leghorn Chickens" IBT #8746, have been located in Bio-Test's freezer in Chicago. Bio-Test is presently identifying these samples with our matrix numbers and expects to be able to ship these samples to us the week of 12-16-71.

Tissue samples from the "90 Day Subacute Oral Toxicity Studies" with Aroclors 1016, 1221, and 5442 in dogs and with Aroclors 1016, 1221, 5432, and 5442 in rats have been received at South Second Street. Tissues from the 90 day study with Aroclor 5432 in dogs are to be shipped by Bio-Test as soon as possible. Tissues from the remaining 90 day studies with MCS 1109 in both beagle dogs and rats will not be available before 2-15-72.

The tissues in hand from the animals fed PCB's are being analyzed at this time.

The tissues from the animals fed PCT's are awaiting extension of the PCT methodology to biological samples (recovery data). Tissues from all remaining studies will not be available before 1-15-72 at the earliest and in some cases not until 2-15-72.

FUTURE PLANS

Analysis of selected tissue samples for the level and type of PCB residues retained from the following feeding studies carried out by Industrial Bio-Test Laboratories.

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PROGRESS REPORT
ORGANIC CHEMICALS DIVISION
TECHNOLOGY DEPARTMENT

FILED BY W. M. Mees W. J. Litschgi E. S. Tucker	FOR October, 1971	DETAILS YES	REPORT NO 3	DATE 10-29-71
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	*W. B. Papageorge	

PROJECT TITLE
 ENVIRONMENTAL ANALYTICAL STUDIES -
 RESEARCH AND DEVELOPMENT, MANUFACTURING AND OUTSIDE SUPPORT STUDIES

PROJECT OBJECTIVE

Identify, locate and assess the magnitude of Aroclor pollution problems, help determine what needs to be done to control pollution sources, verify the effectiveness of proposed pollution control procedures, guide development of recycle and/or disposal methods, and provide collaborative data or legal and regulatory purposes.

SUMMARY

PCB's typical of our Aroclor products have been found at levels below and above recommended guidelines in:

- Coated paper and paper coatings
- Machine oils and plant effluents from one customer manufacturing site
- Fourteen Monsanto Industrial Chemical Company products from four manufacturing sites
- Chicken Chow used as feed by Industrial Bio-Test Laboratories
- Sewer effluents from one Monsanto Plant site
- Tissue turkey extrabts from the U.S.D.A.
- Two competitive products.

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PCT's typical of Aroclor 5432 were found in:

- Plant effluent samples from one customer manufacturing site.

A table showing the sample sources, types, and the level and type of Aroclor found as well as the support objective is presented in the details section.

Additional external and internal support has been provided in the form of:

- An on site methodology review for F. B. Chamberlin (Director of Research and Development) and R. M. Wilkinson (Senior Project Manager) representatives of the Alton Packaging Division, Alton Box Board Co.
- PCB Seminar presentation on analytical methodology for the Research Council of Poultry and Egg Institute of America in Chicago, Ill.
- PCB problem and progress review for T. Katayama (Marketing Manager) and S. Uneno (Product Director) from Mitsubishi Monsanto Chemical Co.
- Preliminary write up of methods for the determination of PCB's in Santoquin and CamHA.
- Evaluation of PCB perchlorination for quantitation of altered PCB residues.
- Analysis of Monsanto and Competitor Aroclor products for polychlorinated dibenzofurans.
- General internal and external analytical consultation.
- Distribution of Aroclor methodology and reference standards.

FUTURE PLANS

PCB Analysis

1. Three to four J. F. Queeny monthly composite sewer effluent samples.
2. Forty-six machine oil and plant effluent samples from Saginaw Grey Iron Casting Plant, Chevrolet Motor Division.
3. Two oil samples skimmed off trade waste settling tanks from Buick Motor Div., General Motors Corp., Flint, Mich. (2 samples).
4. Four coated paper samples from Champion Papers, Hamilton Mill, Hamilton, Ohio (4 samples).
5. Four fluid samples from an imported VW automobile.
6. Five ACL-59 samples from the W.G. Krummrich Plant.
7. Three raw material samples from P.P.G. Industries.
8. Twelve film and paper extracts from St. Regis Paper Co.
9. One fish from a stream associated with a General Electric Plant.
10. Five intra-lab method check samples from a General Electric Plant Laboratory.

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PCT Analysis

1. Ten plant effluent samples from Monsanto's Anniston plant.
2. Saginaw Foundries, Chev. Motor Div. plant effluent samples.

W. J. Litschgi

E. S. Tucker

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TABLE I
CUSTOMER SERVICE

<u>Sample Source</u>	<u>Sample Matrix</u>	<u>No. of Samples</u>	<u>Level Found</u>	<u>Associated Aroclor</u>	<u>Support Objective</u>
General Motors Corp., Buick Div.	Machine oils	11	Per Cent	A-1242	Pollution Control
	Drainage streams around plant	2	PPB	A-1242	Pollution Control
Champion Paper Company	Paper (writing)	3	PPM Per Cent	A-1242 A-5432	Pollution Control
	Paper Coatings (Micron II)	3	PPM Per Cent	A-1242 A-5432	

MANUFACTURING SUPPORT

J. F. Queeny	Sewer effluent composites	5	PPB	A-1242	Pollution Control
	HCl	1	PPB	A-1242	
J. F. Queeny, W. G. Krummrich, Del. River, and Everett	Finished Products	28	N.D.* to PPB	A-1242	

COMPETITIVE PRODUCTS

Ag. Division, Nitro	Santoquin	6	PPB	A-1242	Determination of level and sources of contamination of competitive animal feed additives
	CaMHA	3	PPB	A-1242	

*None Detected

(Cont'd on next page)

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RESEARCH AND DEVELOPMENT

<u>Sample Source</u>	<u>Sample Matrix</u>	<u>No. of Samples</u>	<u>Level Found</u>	<u>Associated Aroclor</u>	<u>Support Objective</u>
Inter-Laboratory Round Robin (WGK, JFQ and Anniston)	Distilled water spiked with known quantities of Aroclor	7	PPB to PPM	A-1242 A-1254 A-1260	Insure the maintenance of precision and accuracy in Monsanto labs analyzing for PCB's
Bio-Test Laboratories, Chicago, Ill.	Ralston-Purina Chicken Chow	2	PPB	A-1242	Determination of PCB back- ground in feed used in the toxicity studies being performed at Bio-Test
USDA	Turkey Extract	1	-	A-1248	Determination of type of PCB and how the PCB contamination occurred

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DETAILS (Cont'd)

Customer ServiceGeneral Motors Corporation - Buick Division

Due to increased pressure from the Michigan State Regulatory Agency concerning PCB releases into the environment, Monsanto was requested to analyze samples of Buick's plant effluent and nearby drainage stream. At the same time, samples of oil used in their presses and machines were submitted to determine residual PCB levels remaining after Buick switched from Pydraul 312 to Pydraul 312A.

<u>Sample Description</u>	<u>Level Found</u>	<u>Aroclor</u>
Water from trade waste prior to Hickory stream	8 PPB 15 PPB	A-1242 A-5432
Water from Hickory rain stream prior to river	13 PPB <5 PPB	A-1242 A-5432
Machine #1	14.8%	A-1242
Machine #2	2.5%	A-1242
Machine #3	13.8%	A-1242
Machine #4	8.9%	A-1242
Machine #5	3.9%	A-1242
Machine #8	4.4%	A-1242
Machine #9	24.3%	A-1242
Trim Press #1	11.0%	A-1242
Trim Press #2	0.9%	A-1242
Trim Press #8	0.8%	A-1242
Trim Press #9	16.8%	A-1242

Champion Paper Company

Coated paper and the used coating (Micron II) samples submitted by Champion Paper Company were analyzed to determine the level of residual PCB's in the PCT's used in the paper coating fluid.

<u>Sample Description</u>	<u>Level Found</u>	<u>Aroclor</u>
Paper - 3866F	203 PPM 0.29%	A-1242 A-5432
Paper - 3878F	140 PPM 0.25%	A-1242 A-5432
Paper - 3758M	217 PPM 0.28%	A-1242 A-5432
Micron II - Sample #1	242 PPM 0.48%	A-1242 A-5432
Micron II - Sample #2	331 PPM 0.75%	A-1242 A-5432
Micron II - Sample #3	211 PPM 0.56%	A-1242 A-5432

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DETAILS (Cont'd)

Manufacturing SupportJ. F. Queeny Plant

<u>Sample Description</u>	<u>Level Found</u>	<u>Aroclor</u>
Clean Acid Sewer (August)	19 PPB	A-1242
DA Sewer (August)	29 PPB	A-1242
MSD Gatewell (August)	77 PPB	A-1242
Clean Acid Sewer (September)	3 PPB	A-1242
MSD Gatewell (September)	31 PPB	A-1242

The DA sewer (August) was supposed to be the Clean Acid Sewer (August) spiked with ~20 PPB A-1242. As can be seen, our results reflect only a 50% recovery. Discussion with the J. F. Queeny pollution group is planned in an effort to resolve this problem.

Monsanto Industrial Chemicals Company

The following Monsanto Industrial Chemicals Company products have been analyzed for PCB's to determine if any of them were significantly contaminated with PCB's.

<u>Sample Description</u>	<u>Level Found</u>	<u>Aroclor</u>
Aspirin QA-1187 9-13-71	N.D.	
Aspirin QA-1213 10-4-71	N.D.	
Maleic Anhydride QA-1295 9-16-71	N.D.	
Maleic Anhydride QA-1333 10-6-71	N.D.	
Sodium Saccharin QA-153 8-31-71	N.D.	
Sodium Saccharin QA-168 9-11-71	N.D.	
Saccharin QA-209 5-5-71	0.03 PPM	A-1242
Saccharin QA-1012 6-23-71	0.02 PPM	A-1242
Vanillin USP Ba#1025	0.20 PPM	A-1242
Vanillin USP Ba#1027	0.18 PPM	A-1242
Tall Oil Rosin Rec'd 1-25-71	N.D.	
Tall Oil Rosin Rec'd 5-25-71	N.D.	
CDP-OR175000 #4	0.17 PPM	A-1242
CDP-04175000 #3 KA-79	0.12 PPM	A-1242
S-334F EUT #A-134 10-4-71	0.26 PPM	A-1242
S-334F EUT #A-135 10-4-71	0.21 PPM	A-1242
S-711 "A" Line 10-6-71	0.27 PPM	A-1242
S-711 "B" Line 10-6-71	0.36 PPM	A-1242
S-160 DA-614	0.36 PPM	A-1242
S-160 DA-803 D.R.	0.35 PPM	A-1242
HB-40 #1 Stg Tk 10-7-71	N.D.	
HB-40 #3 Stg Tk 10-6-71 AA-275C	N.D.	
Biphenyl AA-194 9-27-71	0.67 PPM	A-1242
Biphenyl #2 Stg Tk 10-6-71	0.15 PPM	A-1242
Santowax R 10-6-71	N.D.	
Santowax R Rec'd 9186 10-7-71	N.D.	

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DETAILS (Cont'd)

Competitive Products

<u>Sample Origin</u>	<u>Sample Description</u>	<u>Level Found</u>	<u>Aroclor</u>
Thompson-Hayward Chemical Co.	Ethoxyquin (66.7%)	749 PPB	A-1242
HIC 7-21-71	Neoquin	184 PPB	A-1242
Traveler's Rest	Santoquin Emulsion	151 PPB	A-1242
Traveler's Rest	Santoquin Mix 6	570 PPB	A-1242
Rexolin Chemical	Ethoxyquin (100%)	155 PPB	A-1242
SNS 8-30-71	Abiquin	133 PPB	A-1242

Our detection limit based upon observed background and sample was ~50 PPB.

Roche Ag Lot 2-4-71 Mfgd by Degussa	DL-Methionine	2.5 PPB	A-1242
Upland, Calif. A3NG, SNS	DL-Methionine	21.4 PPB	A-1242
Thompson-Hayward Chemical Co.	DL-Methionine	45 PPB	A-1242

These samples were analyzed to determine if products in competition with Monsanto's Santoquin and CamHA were also contaminated with PCB's. In all cases, they were found to contain an equal or greater level than the corresponding Monsanto products.

Methods for the determination of polychlorinated biphenyls in Santoquin (Organic Research Method 71-26) and CamHA (Organic Research Method 71-27) have been written and will be sent to the Nitro plant where they will be used to monitor their Santoquin and CamHA production.

Research and DevelopmentAnalysis of Chicken Feed from Bio-Test

The chicken feed used in the Bio-Test toxicity studies was purchased from the Ralston-Purina Company. In the past months, attention has focused upon the fact that Ralston's feed has, in some instances, become contaminated with PCB's. For this reason, two samples of the feed used in the chicken feeding studies were analyzed for PCB's.

<u>Sample Origin</u>	<u>Sample Description</u>	<u>Level Found</u>	<u>Aroclor</u>
WCRF-I	Chicken Feed	240 PPB	A-1242
WCRF-II	Chicken Feed	263 PPB	A-1242

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DETAILS (Cont'd)

USDA Turkey Extracts

A USDA extract of a PCB contaminated (Swift) turkey tissue sample was submitted to our laboratory to see if we could establish what original Aroclor product caused the contamination and possibly the type of exposure. Using EC/GC and GC/Mass, we were able to determine that (1) PCB's were really present, (2) Aroclor 1248 was most probably the product involved, and (3) the birds either received a large single dose early in their lives or more probably a lower dose over an extended period of time.

PCT Methodology Status

A method has been developed and presently is being written (Analytical Method #71-37) for analysis of water and SCAS sludge samples for PCT's. Recovery data obtained from samples spiked with A-5432 and 5460 were essentially quantitative at a concentration level of >2 PPM.

This method is currently being used to analyze samples from the biodegradation studies and is at the same time being adapted for use on customer service and tissue samples.

Inter-Plant Round Robin

In order to establish the accuracy and precision of the PCB analysis being carried out in Monsanto laboratories, duplicate sets of spiked water samples have been analyzed by Anniston, W GK and Applied Sciences. The results were as follows:

	PPM PCB (Spiked)			Applied Sciences			W GK		
	A-1242	A-1254	A-1260	A-1242	A-1254	A-1260	A-1242	A-1254	A-1260
1W	-	-	-	-	-	-	-	-	-
2W	0.57	1.04	-	0.74	0.92	-	1.04	1.05	-
3W	0.57	0.10	-	0.86	0.07	-	0.57	0.15	-
4W	5.68	1.04	1.27	1.07	-	-	6.70	-	2.84
5W	5.68	0.52	-	5.25	0.52	-	6.55	1.20	-
6W	-	0.10	-	-	0.06	-	-	0.07	-
7W*	-	-	-	0.92	0.06	-	-	-	-
8W*	-	-	-	-	-	-	8.80	-	4.16

	Anniston		
	A-1242	A-1254	A-1260
1W	-	-	-
2W	0.78	0.75	-
3W	0.45	-	-
4W	5.4	2.95	2.18
5W	9.9	-	-
6W	-	0.03	-
7W*	0.4	0.2	-
8W*	8.4	-	-

*Applied Sciences
7W = 3W

W GK
7W = 1W
8W = 4W

Anniston
7W = 2W
8W = 5W

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The data have been submitted for statistical analyses.

DETAILS (Cont'd)

National Broiler Council Intra-Laboratory PCB Methodology Check

Ten sets of soybean oil and chicken fat were prepared by Monsanto and distributed by Ralston-Purina to government and industrial laboratories analyzing samples for PCB's. The objective of this intra-laboratory check is to establish the validity of the different PCB methods currently being employed.

Listed below are the labs responding, however the individual laboratories were not identified.

	Results PPM					
	1S	2S	3S	4C	5C	6C
Spike Level	0	5.0	2.5	5.0	5.0	2.0
Lab A	0.25	5.31	2.56	4.00	5.06	3.86
Lab B	0.61	3.7	3.0	1.8	4.2	2.1
Lab C (Applied Sciences)	0.78	5.78	3.64	4.13	4.08	3.26
Lab D	#1)0.10	4.17	1.81	2.05	2.05	1.53
	#2)0.10	3.58	1.83	2.18	2.09	1.55
Lab E	0.73	4.40	2.24	2.42	2.68	2.14
Lab F	0.32	5.10	2.91	2.62	2.66	2.22
Lab G	<0.10	2.9	2.0	3.0	3.6	2.9
Lab H	1.10	1.5	<0.6	<0.6	5.5	5.0
Lab I			No Report	Received		
Lab J			No Report	Received		
WGK			No Report	Received		
Anniston	0.38	1.54	0.61	0.57	0.14	-

The results of this inter and intra sample exchange will be submitted for statistical analysis as soon as all laboratories have reported.

Chlorodibenzofurans in PCB's and PCT'sChlorodibenzofurans Found

<u>Sample Description</u>	<u><4Cl/Mol.</u>	<u>4Cl/Mol.</u>	<u>5Cl/Mol.</u>
Prodelec Phenoclor DP6	ND (<2 PPM)	Detected	Detected (15-30 PPM)
Prodelec Phenoclor DP6	ND (<2 PPM)	Detected	Detected (15-30 PPM)
Bayer Clophen A60, Lot 931134	ND (<2 PPM)	Detected	Detected
Kaneclor KC-300 Shizuki, July 1970 (Composition similar to A-1242)	ND (<2 PPM)	ND (<2 PPM)	ND (<2 PPM)
Monsanto A-1242, Lot AK-255	ND (<2 PPM)	ND (<2 PPM)	ND (<2 PPM)
Monsanto A-1254, Lot AK-38	ND (<2 PPM)	ND (<2 PPM)	ND (<2 PPM)
Monsanto A-1260, Lot AK-3	ND (<2 PPM)	ND (<2 PPM)	ND (<2 PPM)
Monsanto MCS-1016, Drum B	ND (<2 PPM)	ND (<2 PPM)	ND (<2 PPM)
Monsanto MCS-1016, KA-708	ND (<2 PPM)	ND (<2 PPM)	ND (<2 PPM)
Monsanto A-5442, Lot AI-9	ND (<5 PPM)	ND (<5 PPM)	ND (<5 PPM)

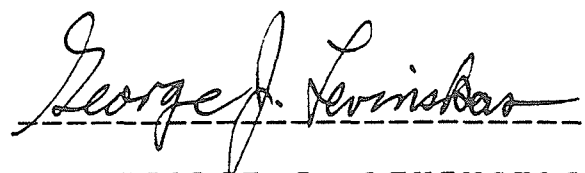
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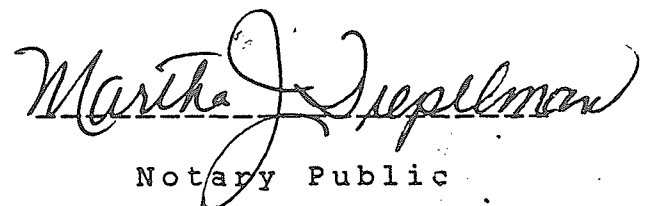
COMES NOW THE WITNESS, GEORGE
J. LEVINSKAS, and having read the foregoing
transcript of the deposition taken on the 2nd
day of November, 1992, acknowledges by
signature hereto that it is a true and
accurate transcript of the testimony given on
the date hereinabove mentioned.



GEORGE J. LEVINSKAS

Subscribed and sworn to before me
this 11th day of December, 1992.

My Commission expires: Aug 4, 1995



Notary Public
MARSHA J. NEPLEMAN
NOTARY PUBLIC STATE OF MISSOURI
ST LOUIS COUNTY
MY COMMISSION EXPIRES AUG 4 1995

ORIGINAL

DEPOSITION CORRECTION SHEET

In Re: *TRANSWESTERN PIPELINE CO. vs. MONSANTO CO.*
CASE NO. BC 026959

Upon reading the deposition and before subscribing thereto,
the deponent indicated the following changes should be made:

Page *10* Line *16* Should read: *POSITION* INSTEAD OF *PHYSICIAN*

Reason assigned for change: *CORRECTION*

Page *19* Line *19* Should read: *WERE THEN GIVEN THE*

Reason assigned for change: *CORRECTION*

Page *25* Line *23* Should read: *BARNARD*

Reason assigned for change: *SPELLING CORRECTION*

Page *39* Line *9* Should read: *FOREIGN* INSTEAD OF *CORN*

Reason assigned for change: *CORRECTION*

Page *41* Line *21* Should read: *WAYLAND* INSTEAD OF *WAYLON*

Reason assigned for change: *SPELLING CORRECTION*

Page *45* Line *23* Should read: *URINARY* INSTEAD OF *URINATED*

Reason assigned for change: *CORRECTION*

Page *70* Line *19* Should read: *DELETE, TO*

Reason assigned for change: *CORRECTION*

Page *74* Lines *5, 6, 19, 22, 23* Should read: *DR. SQUIRE* INSTEAD OF *SQUIRES*

Reason assigned for change: *SPELLING CORRECTION*

Page *74* Line *8* Should read: *SHUBICK* INSTEAD OF *SCHUBIC*

Reason assigned for change: *SPELLING CORRECTION*

George J. Levinson

DEPOSITION CORRECTION SHEET

In Re: **TRANSWESTERN PIPELINE CO. VS. MONSANTO Co.**
CASE NO. BC 026959

Upon reading the deposition and before subscribing thereto,
the deponent indicated the following changes should be made:

Page **74** Line **13** Should read: **POUR INSTEAD OF POOR**

Reason assigned for change: **SPELLING CORRECTION**

Page **75** Line **10** Should read: **SQUIRE'S**

Reason assigned for change:

Page **86** Line **25** Should read: **BODY WEIGHT, THERE WERE CHANGES**
TO ONLY IN FEMALES FED AROCLOR 1242
~~Reason assigned for change:~~ **AND LIVER WEIGHTS ARE AFFECTED**
IN ALL GROUPS EXCEPT MALES FED
AROCLOR 1242.

Page **87** Line **3** Should read: **(ADDED PHRASES UNDERLINED) CLARITY. DISCUSSION WAS ABOUT**
Reason assigned for change: BODY WEIGHT & LIVER CHANGES. THAT
REFLECTS STATEMENTS IN EXHIBIT
1076.

Page **89** Line **3** Should read: **CHANGES INSTEAD OF EXCHANGES**

Reason assigned for change: **CORRECTION**

Page Line Should read:

Reason assigned for change:

Page Line Should read:

Reason assigned for change:

Page Line Should read:

Reason assigned for change:

Page Line Should read:

Reason assigned for change:

George J. Levinthal