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

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| 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.

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## GORE REPORTING COMPANY

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| TRANSWESTERN PIPELINE COMPANY, Plaintiff, | ) | |
| v. | ) | No. BC 026959 |
| MONSANTO COMPANY and DOES 1 through 200, inclusive, Defendants. | ) | |

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

APPEARANCES:

FOR THE PLAINTIFF:

Ms. Dana K. Welch
Shearman & Sterling
21st Floor
725 South Figueroa Street
Los Angeles, California 90017
(213) 239-0300

FOR THE DEFENDANTS:

Mr. Charles F. Preuss
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EXHIBITS

Whereupon. . .

GEORGE J. LEVINSKAS,

of sound mind, having been first duly sworn to tell the truth, the whole truth, and nothing but the truth in the case aforesaid, testified upon his oath as follows, to-wit:

EXAMINATION

QUESTIONS BY MS. WELCH:

Q. Good morning, Mr. Levinskas.

A. Good morning.

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

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

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

A. Yes, I have.

Q. In how many cases?

A. Perhaps a dozen or so.

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

A. Probably half of them.

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

A. Not particularly.

Q. When was the last time you were deposed?

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

Q. Do you recall the name of that case?

A. It was an acrylonitrile lawsuit.

Q. Excuse me?

A. Acrylonitrile.

Q. So that's not related to PCBs?

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

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

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

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

(Brief interruption.)

BY MS. WELCH:

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

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

A. Yes, I do.

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

court of law.

A. That is correct.

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

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

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

A. Wesleyan University.

Q. In what year?

A. 1949.

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

A. The University of Rochester.

Q. In what year?

A. 1953.

Q. When did you start working at Monsanto Company?

A. July 1971.

Q. What was your job position at that point?

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

Q. What were your job responsibilities?

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

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

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

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

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

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

A. That's correct.

Q. So if there were a question about

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

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

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

A. Yes.

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

A. Not specifically.

Q. How about generally?

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

Q. Who did you report to?

A. Mr. Elmer Wheeler was my immediate supervisor.

Q. Did he report to Dr. Kelly?

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

Q. So you were within the auspices of

the Medical Department?

A. Yes.

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

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

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

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

Q. Now, are you speaking about toxicological testing?

A. Yes.

Q. Any other kind of testing?

A. If deemed necessary, it could be

recommendations for other types of testing.

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

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

Q. Are you trained as a toxicologist?

A. My first employment was teaching toxicology.

Q. Would you consider that your area of expertise?

A. Yes.

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

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

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

A. Probably within a few months to relatively soon.

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

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

Q. How did you become aware of PCBs?

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

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

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

BY MS. WELCH:

Q. Go ahead and answer if you understand.

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

referring to?

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

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

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

A. The effects in what systems?

Q. Any systems.

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

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

A. Yes, that would have been

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

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

A. I cannot recall.

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

A. Various scientific journals, technical journals.

Q. Do you recall any of those right now?

A. No, not specifically.

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

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

Q. At the time, were you reading a

scientific journal by the name of "Nature"?

A. Not very often, no.

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

A. Not very often.

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

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

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

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

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

A. Not specifically.

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

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

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

A. I cannot recall that there were.

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

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

Q. Were these chlorinated hydrocarbons?

A. No.

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

A. About a year or two.

Q. What was your next position?

A. It was Manager of Environmental

Assessment and Toxicology.

Q. Did your job responsibilities change?

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

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

A. It was Dr. William Hunt.

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

A. Not that I know of.

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

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

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

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

Q. Go ahead.

A. Shelanski Laboratories.

Q. Could you please spell that?

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

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

A. Industrial Bio-Test.

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

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

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

MR. PREUSS: I'm going to object

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

BY MS. WELCH:

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

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

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

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

could include some environmental aspects.

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

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

Q. And was he responsible for overseeing all toxicology?

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

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

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

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

A. Yes.

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

A. Yes.

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

A. Yes.

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

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

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

A. In about '80 or '81.

Q. And what happened then?

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

Q. How long did you remain in that position?

A. Till about 1986.

Q. What happened then?

A. I was named Senior Toxicology

Consultant.

Q. How long did you remain in that position?

A. September 1991.

Q. And what happened then?

A. I retired.

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

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

Q. Have there been any unofficial contacts?

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

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

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

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

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

(Witness peruses said document.)

A. Okay.

BY MS. WELCH:

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

A. I do not recall seeing this document before.

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

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

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

31, 1971?

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

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

BY MS. WELCH:

Q. You mean the first report?

A. Right.

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

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

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

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

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

A. No.

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

A. Yes.

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

A. I would say probably in the latter Seventies.

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

A. Yes, there was a medical library.

Q. And that was in 1971?

A. Yes.

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

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

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

you?

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

Q. Why was that?

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

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

A. I do not know that.

Q. Who maintained the library in 1971?

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

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

MR. PREUSS: In 1971?

MS. WELCH: Yes.

A. I would have asked one of the

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

BY MS. WELCH:

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

A. No, I do not.

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

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

Q. The second one or the first one?

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

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

A. No, that would have been much later.

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

A. Mid to latter Seventies.

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

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

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

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

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

A. Probably that time.

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

A. I cannot recall.

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

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

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

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

Q. At Industrial Bio-Test?

A. At Industrial Bio-Test.

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

A. I do not recall doing so.

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

A. Yes, I believe I did.

Q. Do you recall approximately when?

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

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

5442 and MCS 1016," dated March 25th, 1971. Did you review that report in 1971?

A. I do not recall that I did so.

Q. Do you recall having reviewed it subsequently?

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

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

A. No, I do not.

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

A. They could be; I do not know.

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

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

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

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

A. Not in 1971.

Q. Do you recall reviewing it subsequently?

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

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

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

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

A. I do not recall looking at any one

of those three reports at any time.

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

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

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

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

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

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

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

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

A. Yes.

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

A. That's correct.

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

A. No, I did not.

Q. Was Mr. Mees a colleague of yours?

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

Q. And he was not in the Medical Department?

A. He was not in the Medical Department.

Q. How about E. S. Tucker?

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

Q. And how about W. J. Litschgi?

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

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

A. I do not recall reviewing that at all.

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

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

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

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

BY MS. WELCH:

Q. You can answer.

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

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

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

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

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

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

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

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

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

you.

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

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

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

A. That's an observation, yes.

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

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

Q. Would you classify that as an adverse effect?

A. I think it can be adverse or beneficial.

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

A. I don't know which to expect in

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

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

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

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

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

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

introduced into an animal.

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

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

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

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

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

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

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

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

(Witness peruses said document.)

BY MS. WELCH:

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

A. Yes.

Q. Is this a document that you prepared?

A. Yes.

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

A. Yes.

Q. At or around October 13th, 1971?

A. Yes.

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

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

Q. Do you recall preparing this document?

A. Yes.

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

A. Yes, in general.

Q. So you know who Dr. Kimbrough is?

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

Q. How did you know her?

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

the line, he introduced me to Dr. Kimbrough.

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

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

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

A. In very general terms, yes.

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

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

MS. WELCH: That's correct.

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

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

Q. Did she contact you?

A. No, I called her.

Q. Do you recall why you called her?

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

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

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

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

A. Not with Dr. Kimbrough.

Q. With anyone else?

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

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

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

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

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

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

Q. And you knew that at the time?

A. Yes.

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

A. It's an excretion of porphyrins in

the urine.

Q. And what are porphyrins?

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

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

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

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

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

Q. And what could cause porphyria?

A. A variety of things.

Q. Can you give me some examples?

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

Q. Was it a surprise to you to hear

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

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

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

A. I said not particularly.

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

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

A. I do not know that there were

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

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

A. I, I cannot specifically recall.

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

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

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

A. No.

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

A. No.

Q. Did you have any discussions with

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

A. No.

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

A. Yes.

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

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

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

A. I do not.

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

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

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

A. Our offices were in close

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

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

A. No, I would not have expected to.

Q. Why is that?

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

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

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

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

yes.

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

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

MS. WELCH: I'm introducing Exhibit 1075.

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

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

(Witness peruses said document.)

(Witness peruses said document.)

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

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

MS. WELCH: Oh, sure.

BY MS. WELCH:

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

A. Yes.

Q. And you recall authoring this report?

A. Yes, I do.

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

A. Yes.

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

A. I considered it so.

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

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

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

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

and references and putting it together.

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

A. Yes.

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

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

Q. Do you recall this as a complete

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

A. No, this was --

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

MS. WELCH: Studies had been done in general.

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

BY MR. WELCH:

Q. With respect to --

A. The three?

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

A. That's correct.

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

A. Yes.

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

A. Yes.

Q. Three-generation rat reproduction study?

A. Yes.

Q. A rat teratology study?

A. Yes.

Q. What is teratology?

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

Q. A dominant lethal mutagenic study in mice?

A. Yes.

Q. And what is lethal mutagenic

study?

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

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

A. Yes.

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

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

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

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

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

BY MS. WELCH:

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

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

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

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

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

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

Q. Well, let me phrase it in this

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

A. Yes.

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

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

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

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

Q. That was in the mid Fifties?

A. I got there in 1958.

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

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

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

Q. That was the early 1960's?

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

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

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

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

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

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

A. That would have been done, yes.

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

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

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

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

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

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

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

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

A. Yes.

Q. Am I pronouncing that right?

A. Sigh-AN-a-mid.

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

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

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

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

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

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

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

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

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

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

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

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

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

Q. Was that available to people at Monsanto?

A. Yes. It's available to everybody.

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

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

reputable.

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

A. Yes.

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

A. Yes.

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

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

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

difficult to conduct a two-year study.

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

A. Not knowingly.

Q. Not intentional toxicological experiments?

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

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

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

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

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

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

and they should be able to tolerate this.

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

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

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

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

A. "-- hepatocytes."

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

A. Hypertrophy.

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

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

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

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

A. High-PER-truh-fee.

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

A. Yes.

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

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

MR. PREUSS: That's the preceding page.

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

BY MS. WELCH:

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

A. Yes.

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

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

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

A. That's right.

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

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

(Witness peruses document.)

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

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

differences from what they saw the first time.

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

A. Right.

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

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

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

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

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

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

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

(Discussion off the record.)

BY MS. WELCH:

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

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

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

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

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

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

1260.

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

BY MS. WELCH:

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

A. Yes.

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

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

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

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

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

MS. WELCH: I think he testified

that he reviewed both sets of slides.

MR. PREUSS: You misunderstood him.

A. No.

MR. PREUSS:

MR. PREUSS: Dr. Pour, I think.

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

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

MR. PREUSS: I'll object. That

mischaracterizes what he said.

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

BY MS. WELCH:

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

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

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

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

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

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

Q. Was she speaking specifically of carcinogenic harm?

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

Q. And what article is this?

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

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

A. I, I mean she's presented her

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

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

(Recess)

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

BY MS. WELCH:

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

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

BY MS. WELCH:

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

(Witness peruses said document.)

A. All right.

BY MS. WELCH:

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

A. No, they are not.

Q. Do you know whose initials those are?

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

Q. Did you author this document?

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

Q. Do you recall authoring this document?

A. Not specifically.

Q. But you believe that you did?

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

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

business activities?

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

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

A. Yes.

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

A. No.

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

A. Yes.

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

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

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

A. That's correct.

Q. In dogs and rats in that feeding study.

A. Right.

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

A. Yes.

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

A. Yes.

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

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

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

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

A. That's right.

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

A. Yes.

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

A. Yes.

Q. Do you know what is acute toxicity mean?

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

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

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

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

A. I was told that by others.

Q. Do you know who told you that?

A. Not specifically.

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

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

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

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

Q. That started in 1969?

A. Somewhere about that there.

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

A. These would be the same tests.

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

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

BY MS. WELCH:

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

MR. PREUSS: Right.

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

MR. PREUSS: All right.

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

MR. PREUSS: You want him just to

read it?

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

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

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

BY MS. WELCH:

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

MR. PREUSS: Female rats.

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

MR. PREUSS: Lifetime rat feeding

studies.

MS. WELCH: We understand it's rats.

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

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

BY MS. WELCH:

Q. Is that correct?

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

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

A. Yes.

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

A. Yes.

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

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

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

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

A. Yes.

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

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

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

cholangiohepatomas; is that correct?

A. Yes.

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

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

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

A. Yes.

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

A. Yes.

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

that sentence means?

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

Q. Would this be an example of enzyme induction?

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

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

A. Yes.

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

A. Yes.

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

A. Yes.

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

A. That's right.

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

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

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

A. Yes.

Q. And what was the effect of the

Aroclor?

A. There was a decreased egg hatchability.

Q. What does that mean?

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

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

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

Q. Any other effects?

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

The young chicks did not survive as well.

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

A. Yes.

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

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

A. Yes.

Q. Is fatty tissue where the PCBs are accumulated?

A. Yes, similar to other chlorinated hydrocarbons.

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

A. One would anticipate that as a possibility.

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

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

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

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

(Witness peruses said document.)

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

(Witness peruses said document.)

THE WITNESS: All right.

BY MS. WELCH:

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

A. I do not recall seeing this document before.

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

A. I could have received some, yes.

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

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

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

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

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

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

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

the tissue? For Aroclor 1242, as an example?

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

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

A. Yes.

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

A. Yes.

Q. And so on.

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

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

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

A. No.

MS. WELCH: I introduce this as Exhibit 1078.

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

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

MS. WELCH: You are right.

BY MS. WELCH:

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

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

BY MS. WELCH:

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

Please take a few minutes to

review this document.

(Witness peruses said document.)

BY MS. WELCH:

Q. Have you ever seen this document before?

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

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

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

Q. How about the environmental analytical program?

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

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

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

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

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

Q. And this refers to the beagle dogs?

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

Q. I believe the other was chickens.

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

Q. The statement is made on the second page, "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." Were you aware of this fact with reference to dogs?

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

somewhat similar comment.

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

A. I do not know why those were chosen.

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

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

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

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

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

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

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

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

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

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

(Plaintiff's Deposition Exhibit 1079 marked for

identification.)

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

(Witness peruses said document.)

BY MS. WELCH:

Q. Do you recall having seen this document before?

A. I do not recall having seen it.

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

A. I do not recall.

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

A. I have never analyzed any PCB effluents.

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

A. I do not recall hearing of it.

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

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

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

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

MS. WELCH: Yes.

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

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

STATE OF MISSOURI )

SS: )

CITY OF ST. LOUIS )

I J. Bryan Jordan, notary public in and for the State of Missouri, duly commissioned, qualified and authorized to administer oaths and to certify depositions, do hereby certify that pursuant to agreement in the civil cause now pending and undetermined in the Superior Court of the State of California, to be used in the trial of said cause in said court, I was attended at the offices of Bryan Cave, in the City of St. Louis, State of Missouri, by the aforesaid witness and by the aforesaid attorneys, on the 2nd day of November, 1992.

The said witness, being of sound mind and being by me first carefully examined and duly cautioned and sworn to testify the truth, the whole truth, and nothing but the truth in the case aforesaid, thereupon testified as is shown in the foregoing transcript, said testimony being by me reported in shorthand and caused to be transcribed into typewriting, and that the

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

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

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

My commission expires July 20, 1994.

J. Bryan Jordan

Notary Public in and for the

State of Missouri

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

GEORGE J. LEVINSKAS

Subscribed and sworn to before me this 11th day of December, 1992.

My Commission expires: Aug 4, 1995

Martha J. Tepelman

Notary Public

MARTHA J. TEPELMAN
NOTARY PUBLIC STATE OF MISSOURI
ST LOUIS COUNTY
MY COMMISSION EXP. AUG 4 1995

ORIGINAL

DEPOSITION CORRECTION SHEET

In Re: TRANSWESTERN PIPELINE CO. VS. MONSANTO CO.
CASE NO. BC026959

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 WYALON

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: SHUBICH INSTEAD OF SCHUBIC

Reason assigned for change: SPELLING CORRECTION

George J. Lewinski

Deponent

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 CHAN
To
~~Reason assigned for change:~~ ONLY IN FEMALES FED AROCLOR 12
AND LIVER WEIGHTS ARE AFFECTE
IN ALL GROUPS EXCEPT MALES FE
AROCLOR 1242.

Page 87 Line 3 Should read: (ADDED PHRASES UNDERLINED) CLARITY. DISCUSSION WAS ABOU
BODY WEIGHT & LIVER CHANGES. TH

Reason assigned for change: REFLECTS STATEMENTS IN EXHIBI
1076.

Page 89 Line 3 Should read: CHANGES INSTEAD OF EXCHANG

Reason assigned for change: CORRECTION

Page Line Should read:

Reason assigned for change:

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Reason assigned for change:

Page Line Should read:

Reason assigned for change:

George J. Lewinski
Deponent

# 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.

## 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 ($LD_{50}$) of Aroclor 1254 for Rats, dated November 10, 1953.

2. The Acute Oral Toxicity ($LD_{50}$) 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

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 28, 1972. IBT No. B9888.

* 7. Toxicity, Reproduction And Residue Study With Aroclor 1221 In White Leghorn Chickens, June 20, 1972. IBT No. J900.

BIOLOGICAL CONSULTANTS
Royal D. Suttkus, 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 ($LD_{50}$) 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 $LD_{50}$ (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 $LD_{50}$ (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 Rosidues 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

REFERENCE:

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

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 diagnoaed 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 apparrently how Weissburg learned about them.

(cont'd.)

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

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Monsanto TECHNOLOGY

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

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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 1260[2] 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 agencies[3]. 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 Ford[4]. 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 rats[6], weanling animals of the Charles River CD strain[7] 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 "Extra[3]" 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.

## References


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.

3. Clutterback, D. (1981). What causes environmental conflict? New Scientist 90, 176-177.

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11. Jenkins, D.H., Keplinger, M.L., Fancher, O.E., Wheeler, E.P. and Calandra, J.C. (1972). Reproductive effects of polychlorinated biphenyls in white leghorn chickens. Toxicol. Appl. Pharmacol. 22, 316.

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15. Monsanto Company (1980). Polychlorinated biphenyls. PCB's. A report on Uses, Environmental and Health Effects and Disposal.

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17. Nelson, N. (1972a). Comments on research needs. Environ. Health Perspect., 1, 181-185.

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19. Pour, P. (1975). Histopathological reevaluation of livers for rats treated with Aroclor. August 1 (unpublished observations).

20. Roberts, J.R., Rodgers, D.W., Bailey, J.A., Rorke, M.A. (1978). Polychlorinated Biphenyls: Biological criteria for an assessment of their effects on environmental quality. National Research Council of Canada. Ottawa. Publication No. NRCC 16077.

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 |
| Findings | | | | | | | | | | | | | | | | |
| 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.

[c] 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 |
| Findings | | | | | | | | | | | | | | | | |
| 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 |
| Findings | | | | | | | | | | | | | | | | |
| 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.

# Toxicity and Environmental Effects of Commercial PCBs

## Introduction

Polychlorinated biphenyls (PCBs) are not a single chemical entity. They are produced by chlorinating biphenyl to acheive 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

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

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 lipoid 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

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 µg/kg/day. It is higher than the safe human intake (1 µg/kg/day) estimated by FDA from human data when it promulgated its tolerances for PCBs in food.

GJL

SCM 019643

## 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 mg ÷ 50 kg ÷ 50 days = 200 µg/kg/day (effect level)
Using 10-fold safety factor leads to estimate that 20 µg/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 µg/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 mg ÷ 365 days = 0.5 mg/day which produced effects.
Assuming 5 kg primate, 500 µg ÷ 5 kg = 100 µg/kg/day
Conclusions: Primate study done at dosages (100 µg/kg/day) greater than the safe dosage (1 µg/kg/day) esimated from human data.
Primate study does show effects at lower dosages over longer time span (100 µg/kg/365 days) than had been observed in man (200 µg/kg/50 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 µg/kg/day based on dog data and 3 µg/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 µg/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.

## PCB Primate and Human Residue Data

| | Primates | | Human |
|---|---|---|---|
| Conc. in diet, ppm | 25 | 2.5 | - |
| Intake, µg/kg/day | 800 | 100 | - |
| Estimated safe dose, µg/kg/day | | - | -/ |
| Liver conc., µg/g | 56.3 | (0.01)* | - |
| Fat conc., µg/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

SCM 019645

000072

# PROGRESS REPORT
ORGANIC CHEMICALS DIVISION
TECHNOLOGY DEPARTMENT

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



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E. S. Tucker/W. M. Mees/W. J. Litschgi

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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) stained 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.

TRAN 022439

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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.

JTURE 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.

| | Tissues Available 2-15-72 |
|---|---|
| 90-Day Subacute Oral Toxicity Study of MCS 1109 with Beagle Dogs | |
| 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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TRAN 022441

# PROGRESS REPORT
## ORGANIC CHEMICALS DIVISION
### TECHNOLOGY DEPARTMENT

| WRITTEN BY | FOR | DETAILS | REPORT NO. | DATE |
|---|---|---|---|---|
| W. M. Mees<br>W. J. Litschgi<br>J. D. Hinchen<br>E. S. Tucker | November-December 1971 | NO | 4 | 11-30-71 |

| LOCATION | JOB NOS |
|---|---|
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E. P. Wheeler/G. Levinskas
E. S. Tucker/W. M. Mees/W. J. Litschgi

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 addition 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 |



TRAN 022437

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.

# PROGRESS REPORT
ORGANIC CHEMICALS DIVISION
TECHNOLOGY DEPARTMENT

| REPORTED BY | FOR | DETAILS | REPORT NO | DATE |
|---|---|---|---|---|
| W. M. Mees<br>W. J. Litschgi<br>E. S. Tucker | October 1971 | YES | 3 | 10-29-71 |

| LOCATION | JOB NOS |
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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 extracts from the U.S.D.A.
- Two competitive products.

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TRAN 022427

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 poly-chlorinated 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.

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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TRAN 022429

TABLE I

CUSTOMER SERVICE

| Sample Source | Sample Matrix | No. of Samples | Level Found | Associated Aroclor | Support Objective |
|---|---|---|---|---|---|
| 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<br>Per Cent | A-1242<br>A-5432 | Pollution Control |
| | Paper Coatings (Micron II) | 3 | PPM<br>Per Cent | A-1242<br>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)

RESEARCH AND DEVELOPMENT

| Sample Source | Sample Matrix | No. of Samples | Level Found | Associated Aroclor | Support Objective |
|---|---|---|---|---|---|
| Inter-Laboratory Round Robin (WGK, JFQ and Anniston) | Distilled water spiked with known quantitites of Aroclor | 7 | PPB to PPM | A-1242<br>A-1254<br>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 background 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 |

DETAILS (Cont'd)

Customer Service

General 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.

| Sample Description | Level Found | Aroclor |
|---|---|---|
| Water from trade waste prior to Hickory stream | 8 PPB | A-1242 |
| | 15 PPB | A-5432 |
| Water from Hickory rain stream prior to river | 13 PPB | A-1242 |
| | <5 PPB | 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.

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

DETAILS (Cont'd)

Manufacturing Support

J. F. Queeny Plant

| Sample Description | Level Found | Aroclor |
|---|---|---|
| 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.

| Sample Description | Level Found | Aroclor |
|---|---|---|
| Aspirin QA-1187 9-13-71 | N.D. | |
| Aspirin QA-1213 10-4-71 | N.D. | |
| Malcic 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. | |

DETAILS (Cont'd)

Competitive Products

| Sample Origin | Sample Description | Level Found | Aroclor |
|---|---|---|---|
| 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 Development

Analysis 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.

| Sample Origin | Sample Description | Level Found | Aroclor |
|---|---|---|---|
| WCRF-1 | Chicken Feed | 240 PPB | A-1242 |
| WCRF-II | Chicken Feed | 263 PPB | A-1242 |

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, WGK and Applied Sciences. The results were as follows:

| | PPM PCB (Spiked) | | | Applied Sciences | | | WGK | | |
|---|---|---|---|---|---|---|---|---|---|
| | 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

WGK
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's

Chlorodibenzofurans Found

| Sample Description | <4Cl/Mol. | 4Cl/Mol. | 5Cl/Mol. |
|---|---|---|---|
| Prodelec Phenoclor DP6 | ND(<2 PPM) | Detected | Detected(15-30PPM |
| Prodelec Phenoclor DP6 | ND(<2 PPM) | Detected | Detected(15-30PPM |
| 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) |