

STATE OF MICHIGAN

IN THE CIRCUIT COURT FOR THE COUNTY OF WAYNE

HOWARD HENDERSON and
MATTIE HENDERSON,

Plaintiffs,

No. 83-330644 NP

JOSEPH WRIGHT and
MATTIE RUTH WRIGHT,

Plaintiffs,

No. 84-424230 NP
(By Consolidation)

CHARLES D. WISE and
ANN ELIZABETH WISE,

Plaintiffs,

No. 84-424231 NP
(By Consolidation)

IRA DAVIS and BERTHA DAVIS,

Plaintiffs,

No. 84-424232 NP
(By Consolidation)

ROBERT NICHOLS and
CAROLYN NICHOLS,

Plaintiffs,

No. 84-427471 NP
(By Consolidation)

-v-

MONSANTO COMPANY, a corporation,
DETROIT EDISON COMPANY, a
corporation, TRANSFORMER INSPECTION
RETROFILL CORPORATION, and GENERAL
ELECTRIC COMPANY, jointly and
severally,

Defendants.

THE DEPOSITION OF WILLIAM B. PAPAGEORGE

reported on Wednesday, May 27, 1987, at 216 North
Meramec, Clayton, Missouri, at or about 10:50 a.m.

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On behalf of the Plaintiffs.

On behalf of Monsanto Company.

On behalf of Transformer Inspection
Retrofill Corporation.

On behalf of General Electric Company.

On behalf of Transformer Inspection
Retrofill Corporation.

LORI B. RESNICK, CSMR-3236

WATER PCB-SD0000058767

I N D E X

WITNESS:

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WILLIAM B. PAPAGEORGE

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C & L Reporting

CERTIFIED COURT REPORTERS

29555 NORTHWESTERN Highway • Suite 508

Southfield, MI 48034

(313) 827-8589

1 Clayton, Missouri

2 Wednesday, May 27, 1987

3 (At about 10:50 a.m.)

4 WILLIAM B. PAPAGEORGE

5 having been sworn to tell the truth, the whole
6 truth and nothing but the truth, was examined and
7 testified upon his oath as follows:

8 MR. LEDBETTER: Let the record
9 reflect that this deposition is being taken
10 pursuant to notice to all parties and will be used
11 for all purposes applicable under the Michigan
12 Court Rules.

13 EXAMINATION

14 BY MR. LEDBETTER:

15 Q Sir, would you state your full name for the
16 record, please?

17 A William B. Papageorge.

18 Q Mr. Papageorge, what's your date of birth?

19 A September 7th, 1922.

20 Q And your current home address?

21 I have a last known one here on
22 your CV on the second page. Is that correct?

23 A That's correct.

24 Q That's 321 Pebble Valley Drive, St. Louis,

1 Missouri, 63141.

2 A That's correct.

3 Q Mr. Papageorge, my name is J.D. Ledbetter. I
4 represent several clients in regard to some
5 allegations of PCB exposure from certain
6 transformers in the Detroit area.

7 I assume you have given depositions
8 before, correct?

9 A Yes.

10 Q So, you're aware of the procedures pretty much
11 overall?

12 A Yes.

13 Q Mr. Papageorge, I've been provided a copy of your
14 CV and I notice the date at the bottom right is
15 6-8-78.

16 Is that current, other than that
17 date perhaps?

18 A There are two additional assignments since that
19 was prepared.

20 Q Okay. Is that additions to the Monsanto
21 experience?

22 A Yes.

23 Q Okay. And what are those?

24 A Starting in 1983, I was a Director of

1 Environmental Operations for Monsanto Industrial
2 Chemicals Company.

3 Q Okay. And how long did you remain in that
4 position? 1983 to what?

5 A That terminated in December, 1985.

6 Q Okay. And then the next correction?

7 MR. SCOTT: Addition.

8 MR. LEDBETTER: Addition,
9 correction, deletion.

10 THE WITNESS: The next assignment
11 was manager of Occupational Health for Monsanto
12 Chemical Company in the calendar year 1986.

13 BY MR. LEDBETTER:

14 Q Okay. Very well. Let me develop some of these a
15 little bit more.

16 What is your current occupation?

17 A I'm self-employed.

18 Q Doing what?

19 A Consulting.

20 Q What type of consulting do you do?

21 A Matters relating to PCBs.

22 Q Do you have a company name?

23 A No.

24 Q Just in your own capacity; William Papageorge?

1 A Correct.

2 Q And you've been doing that since end of '86?

3 A Yes.

4 Q Were you doing any consulting prior to the end of
5 the calendar year 1986?

6 A No.

7 Q Okay. So, we're talking basically five months
8 or so that you've been doing consulting business?

9 A Yes.

10 Q You indicated that the type of consulting you do
11 is relating to PCB matters.

12 Is it all matters concerning PCBs
13 or is it limited to certain categories?

14 A Oh, it's general in nature.

15 It does relate to the manufacture
16 of PCBs, the past uses of PCBs, some of the
17 chemistry of PCBs, some of the industrial
18 hygiene and exposure type information,
19 environmental matters.

20 I believe that covers it.

21 Q How many files do you currently have ongoing that
22 you're involved with --

23 A I'm sorry?

24 Q -- on a consulting basis?

1 How many files do you currently have?

2 A I don't have any files.

3 Q How many clients do you currently have?

4 A One.

5 Q And who is that client?

6 A Smith, Helms, Muscle & Moore (phonetic). They're a
7 a law firm in Greensboro, North Carolina.

8 Q And is that working for the defendant in the case
9 or working for the plaintiff; if you know?

10 A The defendant.

11 Q Do you know who the defendant is in that case?

12 A Monsanto Company.

13 Q Okay. And as far as the consulting business,
14 that's the only file that you've had since -- or
15 the only one you have currently?

16 A Correct.

17 Q Have you had any others in 1987?

18 A No.

19 Q So, just the one?

20 A Yes.

21 MR. SCOTT: Counsel, don't let me
22 mislead you.

23 Don't assume that he has one file
24 because he consults with this law firm. I don't

1 want you to be misled by that.

2 MR. LEDBETTER: Okay. I

3 understand.

4 BY MR. LEDBETTER:

5 Q There's just one matter that you're consulting
6 with, but it could be multiple situations, multiple
7 plaintiffs?

8 A Yes.

9 Q Okay. Now, in your educational background, I
10 noticed you've got a B.S. in Chemical
11 Engineering from Washington University in 1943.

12 A That's correct.

13 Q What years did you attend Washington University?

14 A 1940 to 1943; 1946 to 1947.

15 Q And both of those sessions resulted in degrees;
16 the first one the B.S. in Chemical Engineering?

17 A Yes.

18 Q And the second was an M.S. in Chemical
19 Engineering?

20 A Yes.

21 Q Any other formal education other than those two?

22 A I took courses with -- at that time it was known as
23 Oklahoma A & M; I believe today it's called Oklahoma
24 State -- with the intent of working toward a

1 doctorate degree.

2 Q How many hours did you complete at Oklahoma
3 A & M?

4 A I don't recall specifically.

5 I think it's somewhere about 12
6 hours.

7 Q And you never got to a point of doing a
8 dissertation?

9 A No.

10 Q What years did you attend Oklahoma A & M?

11 A That was during the period 1947 to 1951.

12 Q And, certainly, that time period was not all
13 regular attendance; it had to be sporadic
14 attendance in that four-year period?

15 A Correct.

16 Q Prior to first attending Washington State in
17 1940, were you working or have any other formal
18 education experience above high school level or
19 was that right out of high school?

20 A Out of high school.

21 Q And the period that you were at Washington from
22 1940 to 1943, did you work or just attend
23 school?

24 A I worked in a supermarket in the early years. I

1 have forgotten the exact period of time.

2 Then I was involved with a
3 student aide program at the university, where I was
4 paid by the hour for performing duties in the
5 laboratory. I believe it was called the
6 National Youth Administration at that time.

7 Q Then after 1943, did you begin full-time
8 employment?

9 A No. I was in the military.

10 Q What branch?

11 A Army.

12 Q What did you do in the Army?

13 A Well, I started out in anti-aircraft artillery,
14 spent some time with the infantry, and ended up
15 in the Corps of Engineers.

16 Q How long were you in the Army?

17 A Until 1946.

18 Q Were you discharged honorably at that time?

19 A Yes.

20 Q What was your highest rank?

21 A Captain.

22 Q And in 1946, did you return to school full-time?

23 A Yes.

24 Q And you received your M.S. in '47?

1 A Yes.

2 Q Did you work anywhere in that time period?

3 A No.

4 Q After 1947, where did you begin work?

5 A With the Phillips Petroleum Company in

6 Bartlesville, Oklahoma.

7 Q Bartlesville?

8 A Bartlesville, yes.

9 Q What did you do with Phillips Petroleum in 1947?

10 A I initially worked on problems associated with

11 drilling oil wells and secondary recovery in

12 spent oil fields.

13 Q What do you mean "secondary recovery in spent?"

14 A These are techniques that are used in oil fields

15 that cannot deliver the oil under their own

16 natural means. And we could apply gas, water, or

17 treated waters, to push the water to the surface.

18 Q How long did you work for Phillips Petroleum?

19 A Four years.

20 Q Till 1951?

21 A '51.

22 Q Did you remain generally in the same job duties

23 and responsibilities in that four-year period?

24 A No. The latter assignment involved designing of

1 refinery equipment.

2 Q What kind of refinery equipment did you design?

3 A Tanks and distillation columns.

4 Q Is that designing the equipment from scratch?

5 A Design from scratch.

6 Q Did you work around the electrical industry or

7 electrical equipment at that time period?

8 A No.

9 Q Did you have any involvement with PCBs in that

10 time period?

11 A No.

12 Q Did you ever hear of PCB in that time period?

13 A No.

14 Q Then in 1951, you began working with Monsanto?

15 A Yes.

16 Q In the capacity of chemical engineer?

17 A Yes.

18 Q Where was the J.F. Queeny Plant?

19 A It's located in St. Louis, Missouri.

20 Q Is that plant still in operation?

21 A Yes.

22 Q Okay. As chemical engineer, what did you do in

23 that time period, '51 through '55?

24 A Designing equipment for the manufacture of a

1 chemical.

2 Q What chemical was it?

3 A Phthalic anhydride, p-h-t-h-a-l-i-c; anhydride,
4 a-n-h-y-d-r-i-d-e.

5 Q Can you tell me what that is, roughly, in lay
6 terms?

7 I don't want to know the specific
8 formula. I think I already know more than I
9 need to.

10 MR. SCOTT: Didn't you take
11 chemistry?

12 MR. LEDBETTER: Just a little.
13 but not that much.

14 THE WITNESS: This is a chemical
15 that's used as a raw material in the manufacture
16 of coatings and plastic and plasticizers.

17 BY MR. LEDBETTER:

18 Q Now, as a chemical engineer, you didn't actually
19 work on the chemical itself. You designed the
20 equipment that manufactured the chemical.

21 Is that true?

22 A Designed some of the equipment that was involved
23 in the manufacture of this chemical, yes.

24 Q Okay. Did that job duty remain about the same

1 in that specific equipment, and that specific
2 chemical in that four-year period of '51 to '55?

3 A Yes.

4 Q What were the plasticizers used for; the coating
5 plasticizers that you mentioned?

6 A We -- I didn't mean to imply that we made
7 coatings and plasticizers. We made the
8 phthalic anhydride that was shipped to customers
9 who in turn made these.

10 Q Okay. Do you know generally what applications
11 or uses were intended for that particular
12 chemical?

13 A Well, I can recall some.

14 Q Okay. What are those?

15 A Paints, for example. Resins, such as those used
16 in Fiberglas boat hulls.

17 Plasticizers are materials added
18 to plastics to make them pliable, soft, so they
19 aren't brittle and breakable.

20 Those are the general uses I
21 recall.

22 Q Do you recall whether that particular chemical
23 was toxic?

24 A All chemicals are toxic.

1 Q And it depends on the amount and degree, doesn't
2 it?

3 MR. SCOTT: To what?

4 MR. LEDBETTER: To be toxic.

5 MR. SCOTT: Counsel, I don't
6 understand your question. It's vague.

7 BY MR. LEDBETTER:

8 Q Toxicity depends upon the amount of exposure and the
9 degree of exposure?

10 A And the creature exposed.

11 Q Okay. And that's generally an analysis of
12 toxicity, is it not?

13 MR. SCOTT: What's generally an
14 analysis of toxicity?

15 MR. LEDBETTER: My last question;
16 the degree, the amount, the length of exposure
17 and the creature to be exposed, as Mr. Papageorge
18 added.

19 THE WITNESS: Well, I'm a little
20 confused, but I'd like to suggest that the
21 effect has to be described as well, to understand
22 what is meant by toxicity.

23

24

1 BY MR. LEDBETTER:

2 Q Okay. What do you mean by the effect?

3 A Does it cause death, does it cause incapacities
4 of other kinds?

5 Does it impair functioning of the
6 creature, its ability to reproduce?

7 Many effects are possible.

8 Q And the absence of those effects, would that
9 equal no toxicity?

10 A I would prefer to call it no effect.

11 Q Okay. So, in order to be toxic, you're saying
12 that it will have an effect on somebody if we
13 throw in the other factors, too, that we've
14 talked about; the length of exposure, the amount,
15 etcetera?

16 A Yes.

17 Q Okay. Generally speaking -- what was the chemical
18 again; its full name?

19 A Phthalic anhydride.

20 Q How is phthalic anhydride made?

21 A There are two processus that I'm aware of.

22 One is it starts with naphthalene,
23 which is the mothball chemical. And in the
24 presence of a catalyst and oxygen, the

1 naphthalene is converted into phthalic anhydride.

2 The other process involves the use
3 of another starting material, xylene. Under a
4 different catalyst and different conditions, it too
5 can be converted to phthalic anhydride.

6 Q Does xylene contain PCBs?

7 A No.

8 Q Does naphthalene contain PCBs?

9 A No.

10 Q Okay. From the period 1955 forward, you began as
11 production supervisor, also at the Queeny Plant?

12 A Yes.

13 Q And what did you do as a production supervisor?

14 A I supervised the activities associated with the
15 manufacture of two types of chemicals, as I
16 recall, at that period of time.

17 Q What are the two types of chemicals?

18 A Well, the initial assignment was involved with
19 the manufacture of plasticizers.

20 The second assignment involved the
21 manufacture of a group of chemicals referred to
22 as chlorinated nitro benzenes.

23 Q Is there any distinguishing properties of the
24 plasticizer that you manufactured in the '55 to

1 '56 period and the material that was used for
2 plasticizers in the '51 to '55 period; the
3 phthalic anhydride?

4 A The anhydride I referred to that I was involved
5 with in the '51 to '55 period, is a starting
6 material in the manufacture of the
7 plasticizers with which I was involved with in
8 the '55 to '56 period.

9 Q Okay. So you used that same material then to
10 manufacture the chlorinated nitro benzenes?

11 A No. To manufacture the plasticizers.

12 Q Okay. And what was the plasticizers called? Did
13 they have any particular names or categories?

14 A Yes. There were many. I don't claim to recall
15 all of them.

16 There's ethylphthalates,
17 methylphthalate, butylphthalate, octylphthalate.
18 And as I recall, there were combinations of
19 these. And I don't remember specifically, but
20 it's possible to have such a thing as -- I'm not
21 too certain of this, but it is possible to have
22 like ethylbutylphthalate. Instead of dibutyl,
23 it's ethylbutylphthalate.

24 Q Would that suggest, Mr. Papageorge, that the material

1 in the manufacturing process is slightly changed to
2 go from ethyl to methyl to octyl?

3 A Yes.

4 Q How is that change made?

5 A I don't know quite how to answer.

6 Are you talking about the chemical
7 change or the physical change?

8 Q The chemical change to start. Then you end up
9 with a physical property, don't you?

10 A Yes.

11 Q Okay.

12 A Without getting too technical, I'll try.

13 Q Okay. I would appreciate it.

14 A Phthalic anhydride in its makeups offers
15 opportunity for alcohol to attach itself. This
16 is where the ethyl designation comes from; the
17 use of methyl alcohol or the use of butyl alcohol
18 and methyl for methyl alcohol.

19 Each of these alcohols will attach
20 themselves to one of two places available on the
21 phthalic anhydride molecule. As a result, the
22 phthalic anhydride, which starts out as a solid
23 material at room temperature, is converted then
24 to a liquid which is in most cases colorless like

1 water and looks an awful lot like a mineral oil,
2 baby oil kind of consistency.

3 Q Any odor of alcohol?

4 A It's not so much the alcohol, but each of the
5 phthalates has its characteristic odor. It is
6 generally perceived by most of us as being a
7 rather pleasant odor, not necessarily obnoxious.

8 Q Is the odor put in or is it a natural property?

9 A It's an inherent property of the material.

10 Q Do you recall any specific safeguards in handling
11 or utilizing these particular chemicals in their
12 final products, in the manufacturing stage?

13 A I recall a few. I don't recall them all.

14 Of course, alcohols are explosive,
15 so you have to worry about the flammability
16 aspects of it.

17 They do have a solvent action on
18 skin. Too much exposure can cause a reddening of
19 the skin because the skin's natural oils are
20 removed.

21 The phthalic anhydride in itself
22 is -- can be corrosive like an acid; therefore,
23 it is not permitted to be on the skin and should
24 not be.

1 When it is melted, of course, it
2 creates problems for possible thermal burns. It's
3 like having hot candle wax poured on your skin.

4 Other than that, I don't recall
5 any others.

6 Q Any inhalation problems or potential problems?

7 A The inhalation of the fumes, alcohol or the
8 molten phthalic anhydride or even of the hot
9 plasticizer can be a respiratory irritant.

10 And, of course, if it's overdone, it
11 can have symptoms very much like a severe chest cold.

12 Q It affects the lungs?

13 A Yes.

14 Q Does it have a tendency to build up liquid in the lungs
15 in advanced cases or more extreme exposures?

16 A I've never seen one, but I was told by medical
17 people that it's possible --

18 Q Okay.

19 A -- when it's extreme exposures.

20 Q All right. Is the same thing true, as far as
21 safety precautions and such things, for
22 naphthalene; the same sort of risks?

23 A Naphthalene? Generally, yes.

24 Q Anything distinguishing between the ones that

1 you've just mentioned and naphthalene?

2 A Well, naphthalene is not acidic the way phthalic
3 anhydride is, so it would not have the corrosive
4 properties.

5 But it does have the respiratory
6 irritant properties, and also introduces a
7 potential for headaches.

8 Q Okay. Did you ever learn why it causes
9 headaches?

10 A No.

11 Q How about a problem to the skin, if any?

12 A The problem with naphthalene, again, the fine
13 dust can be an irritant.

14 Of course, the molten material can
15 introduce not only the irritant, but also the
16 burns, the thermal effects.

17 Q And that goes without saying. Even if it were
18 hot water, that would be true, correct?

19 A True. Except that these chemicals, many of them
20 do not cool rapidly, so their ability to
21 cause damage because of high temperatures is much
22 more so.

23 MR. SCOTT: Do you have a few
24 naphthalene cases? Is that why you're making

1 this inquiry?

2 Or are you headed in some
3 direction?

4 MR. LEDBETTER: Oh, yes.

5 BY MR. LEDBETTER:

6 Q How about xylene? The same safety precautions,
7 roughly, similar to those in naphthalene?

8 A Xylene is a liquid at room temperature.

9 In fact, it's an ingredient in
10 many gasoline blends. It, too, can cause the
11 headaches. It can cause lung and respiratory
12 effects at fairly high levels. It has a
13 de-oiling effect on skin.

14 That's all I can remember about
15 xylene.

16 Q In this period of 1951 through 1956, any involvement
17 at all in PCBs?

18 A No.

19 Q Or with PCBs?

20 A No.

21 Q All right. Moving on to the next position, that
22 was 1956 to '57, maintenance supervisor?

23 A Yes.

24 Q What did you do as a maintenance supervisor?

1 A I was assigned a team of workers. This team was
2 involved in the revision or construction of
3 facilities, new facilities or revisions of
4 existing facilities; the small projects.
5 Q When you're talking about facilities, are you
6 talking about Monsanto facilities?
7 A Monsanto production facilities.
8 Q This is at the J.F. Queeny Plant?
9 A That is correct.
10 Q Did you do any facilities outside of that plant?
11 A No.
12 Q Okay. What kind of facilities were you involved in
13 in that year period or thereabouts?
14 A Oh, for example, the replacement of a small, old
15 pump with a modern, bigger pump; or the replacement
16 of a leaking tank with a new tank; or the
17 addition of a tank into a unit because the tanks
18 there were not adequate to do the job; the
19 replacement of steel pipe with a stainless steel
20 pipe because of corrosion problems.
21 Q Did you ever get involved in anything with
22 electrical equipment; replacement or revision?
23 A Yes; to a limited degree.
24 Q Any involvement with transformers?

1 A No.

2 Q What types of electrical equipment did you get
3 involved in?

4 A For example, I mentioned the installation of the
5 pump.

6 It would have associated with it an
7 electric motor and it would have its starting
8 devices, whether they were a manual switch or an
9 automatic instrument controlled from some control
10 panel or desk somewhere.

11 That kind of electrical
12 involvement.

13 Q Okay. And in the time period that we've talked
14 about thus far, '51 through '57 when you were a
15 maintenance supervisor, did you remain within
16 basically the same department at Monsanto?

17 A No. Each of these were different departments.

18 I'm assuming we both have the same
19 definition of "department."

20 Q Okay. In fact, I'll accept your definition of
21 "department" as you knew it at the time; whatever
22 that was.

23 What department were you in as a
24 chemical engineer?

1 A At that time, it was called the Plant
2 Investigations Group.
3 Q And then as a production supervisor?
4 A It was the Plasticizers Department, which was
5 called Department 23.
6 Q The terms are synonymous; Department 23 or
7 Plasticizers Department?
8 A Yes. But a particular type of plasticizer.
9 Q Okay.
10 A Phthalates plasticizers.
11 Q Okay. And that was under the Department 23
12 title?
13 A Correct.
14 Q Okay. So, there could be Department 24 or 22
15 maybe; something of that sort?
16 A Oh, yes.
17 Q Okay.
18 A There was also a department A-5, the Chloral Nitro
19 Benzene Department.
20 Q Okay. And that was Department 5?
21 A A-5.
22 Q A-5?
23 A A dash 5.
24 Q Any particular meaning to the letter or number

27

1 designations that you know of?

2 A No. It depended on different areas of
3 management and how they wanted to designate new
4 departments as they were erected.

5 Q Were all of those departments that you've named
6 thus far, were they like part of a larger
7 division at Monsanto at the Queeny plant?

8 A Well, the plant was managed by general
9 superintendents who were given specific
10 departments to manage, based generally on types
11 of chemicals, but tempered an awful lot by
12 workload so that the workload for each was about
13 equal.

14 That's the only subdivision that I
15 can think of.

16 Q Okay. The departments would be assigned to each of
17 the general superintendents?

18 A Correct.

19 Q How many of those were there, if you recall, in
20 that time period?

21 A I believe at that time there were three general
22 superintendents of manufacturing.

23 Q Okay. Did you remain under one general
24 superintendent for all of those positions, or did

1 that change?

2 A That was one general superintendent.

3 Q All right. Do you recall who that fellow was?

4 A Mr. McCracken; William McCracken.

5 Q Okay. After you left as a general supervisor,
6 what was your position?

7 A I'm sorry. General supervisor of which?

8 Q General supervisor of maintenance, I think it
9 was, and then you moved on to another position.

10 I see on the CV here, it's maintenance
11 superintendent?

12 A Yes.

13 Q What were your job duties and responsibilities
14 there as compared to maintenance supervisor?

15 A Well, this represented a promotion.

16 All of the maintenance supervisors
17 in the plant reported to me as the
18 superintendent.

19 Q And you would report to one of the general
20 superintendents?

21 A The general superintendent of maintenance and
22 engineering, who was in addition to the three
23 manufacturing superintendents.

24 Q How many maintenance supervisors were there

1 reporting to you at that time period?

2 A Five or six. I don't remember exactly.

3 Q Was the J.F. Queeny Plant in this time period

4 we've covered thus far, '51 through - now we're

5 into maybe the '57 to '59 period - were they

6 involved at all in the manufacture of PCBs?

7 A No.

8 Q Any PCB involvement up through this time period,

9 1959, that you had even outside of manufacturing?

10 A As maintenance superintendent, I had reporting to

11 me, individuals who were involved with PCBs.

12 Q Was that by department or group or some sort of

13 supervisor?

14 A They were involved with the maintenance of

15 systems that contained PCBs.

16 Q What kind of systems are you referring to?

17 A I have in mind heat transfer systems, compressors.

18 and transformers and capacitors.

19 Q What does heat transfer system mean?

20 A This is a system in which a liquid is heated to

21 high temperatures in a remote location using gas

22 flames, open flames.

23 The hot fluid is then pumped into

24 an area which does not permit the use of open

1 flames, and the heat in that fluid is used to
2 perform some function. For example, to cook a
3 chemical batch and when the heat is dissipated,
4 the cool liquid is returned back to the furnace
5 to be reheated, and it's continually cycled.

6 Q Would it be similar to like a radiator?

7 A Similar to like a car radiator, yes.

8 Q In this time period that you had some of these
9 fellows reporting to you that were involved with
10 PCBs, did you have any guidelines or directives
11 of any sort that you referred to, to guide you as
12 to how they should be handled or what should be
13 done around them?

14 A The guidelines available were those supplied by
15 the equipment manufacturer.

16 Q Okay. When we're talking equipment manufacturer,
17 the heat transfer system or the transformer or
18 the capacitor, or whatever the case may be?

19 A Or the compressor, yes.

20 Q Okay. Who were the suppliers that provided that
21 equipment in that period of time, if you recall?

22 A I don't pretend to recall all of them, no/

23 Q How about the transformers?

24 A The transformers, I recall, General Electric as a

1 supplier, Westinghouse.

2 I'm not certain of the others.

3 Q So, your PCB involvement, as I understand it in
4 this time period, was that that was contained in
5 systems or components that was provided to you at
6 Monsanto by other outside suppliers?

7 A Yes.

8 Q Do you know who the PCB was made by that went
9 into those components or pieces of equipment that
10 were supplied to Monsanto?

11 A It was a Monsanto PCB.

12 Q Okay. Were you aware of any guidelines or
13 regulations from Monsanto regarding PCBs in that
14 time period?

15 MR. SCOTT: What aspect, counsel?

16 MR. LEDBETTER: Anything to tell
17 them how to handle it or supply it or safety
18 regarding it.

19 MR. SCOTT: As it would pertain
20 to the equipment that he was involved with?

21 MR. LEDBETTER: That's right.

22 MR. SCOTT: All right.

23 THE WITNESS: At that time, no.

24 MR. LEDBETTER: Okay.

1 MR. DECKER: It's been a while since
2 we've had a time indicated. Are you still in '57?

3 MR. LEDBETTER: Yes. We're still
4 in '57 through '59.

5 BY MR. LEDBETTER:

6 Q In this period of time, '57 through '59, did you
7 have at your disposal company regulations or
8 directives or that sort of thing to guide you
9 into the handling of the chemicals or
10 precautions regarding chemicals such as PCBs?

11 MR. SCOTT: In what aspect,
12 counsel?

13 Again, are you talking about PCBs
14 that are contained in the transformers? The
15 question is overly broad.

16 MR. LEDBETTER: Just the area
17 that he was involved in in '57 through '59.

18 MR. SCOTT: I thought he just
19 answered that question.

20 MR. LEDBETTER: No. This is
21 regarding documents and regulations.

22 BY MR. LEDBETTER:

23 Q Are you aware of any of those that you had?

24 A Is your question limited to PCB only?

1 Q Well, no.

2 I'm looking for where you'd go for
3 the regulatory guidance or directives to tell you as
4 a superintendent at that time, the precautions
5 or safety precautions or handling precautions
6 for the chemicals including PCBs.

7 MR. SCOTT: That are what? That
8 are in the machinery that he was maintaining?

9 MR. LEDBETTER: That's right; that he's
10 he's having guys report to him on in '57 through '59.

11 MR. SCOTT: Okay. All right.

12 THE WITNESS: When it came to the
13 types of equipment that we discussed earlier, the
14 transformers, capacitors, compressors, heat
15 transfer systems, we would rely, as I said, on
16 the manufacturer's literature.

17 Considering other chemicals
18 present in the plant, my team of workers would
19 rely very heavily on the manufacturing personnel
20 that used the equipment, for guidance regarding
21 the use of protective equipment; proper
22 decontamination before the maintenance personnel
23 would start work; the permission to enter areas;
24 the permission to use flames as in welding; or

1 to disconnect power or connect power.

2 All of that guidance came out of
3 the manufacturing supervision. They, in turn,
4 had available to them documents describing the hazards
5 associated with their equipment and their chemicals.

6 Q Okay. And the documents that you're referring to
7 would have been primarily at their disposal rather
8 than yours?

9 A Correct.

10 Q And if you had questions of that sort, you would go to
11 manufacturing supervision and ask them for whatever
12 you needed?

13 A That is correct.

14 Q Okay. Do you know where those documents were
15 kept at that period of time?

16 A Well, there's two types, generally.

17 One is the highly-technical document
18 generally referred to as a Standard Manufacturing
19 Process Manual or Document. That would generally
20 be in the supervisor's office.

21 There is another document
22 available which would be referred to as the
23 Standard Operating Instructions or Standard
24 Operating Procedures. These documents were

1 designed for the use of the operating personnel;
2 the people who pushed the buttons and turned the
3 valves and made things happen.

4 They, too, would have sections in them
5 on safety and handling of chemicals and equipment.

6 These copies would be available in the
7 working area, generally in a room referred to as the
8 control room; more or less the center of operations
9 for that particular department.

10 Q Would the standard operating instructions and
11 procedures apply to you in a maintenance function
12 in this time period that we're talking about;
13 '51 through '59?

14 A Certainly.

15 Q Okay. So, those kind of things are something
16 that you would like to have available to you?

17 A They were available to the team involved at that
18 particular time.

19 Q Okay. Do you recall ever reviewing any
20 documents relating to PCBs in that time period?

21 A No.

22 Q Okay. Then in '59, you changed positions again --
23 and I assume again that's a promotion -- to assistant
24 engineering superintendent?

1 A No, that's a lateral move.
2 Q What was your job duties then?
3 A I supervised a team of engineers who were
4 involved with primarily the design of chemical
5 systems, producing systems.
6 Q That's kind of what you did back in 1951 through
7 '55?
8 A That kind of activity. The difference this time
9 is I was responsible for the activities of many
10 as a supervisor.
11 Q And, again, at that time period, you weren't
12 involved in designing the equipment for the
13 production of PCBs?
14 A No.
15 Q And then in 1961, you became general
16 superintendent of warehouse inventory utilities?
17 A Is that a question?
18 Q Yes. I'm sorry.
19 A Yes.
20 Q What were your job duties then?
21 A I was responsible for the receiving of materials
22 of all kinds; the shipping of products, as well
23 as other materials; the operation of the
24 department that distributed electricity, steam

1 and air throughout the plant.

2 In other words, anything that
3 wasn't directly associated with manufacture or
4 maintenance was in this group, service group.

5 Q Okay. Prior to this time, as I understand it,
6 you had been involved under one general
7 superintendent, and now you've got a change where
8 you are a general superintendent?

9 A No, I didn't mean to mislead you.

10 When I was in manufacturing, I
11 was reporting to a general superintendent of
12 manufacturing.

13 Q Okay.

14 A When I went into maintenance, I was reporting to
15 a general superintendent of maintenance and
16 engineering.

17 Now I am the general
18 superintendent of this miscellaneous activities
19 group.

20 Q Okay. So, it's not manufacturing, it's not
21 engineering; it's now called inventories and
22 utilities or something of that sort?

23 A Correct.

24 Q And you're one of the three superintendents that you

1 had mentioned earlier?

2 A No. There's still three manufacturing
3 superintendents.

4 Q Okay. Very well.

5 A And we refer to them as general superintendent
6 of maintenance and engineering and now this is
7 the fifth one.

8 Q All right. In 1961 through '64, did you have
9 any involvement in that time period with PCBs?

10 A Yes.

11 Q In what way?

12 A Two ways.

13 One is as a user of electrical
14 equipment in the electrical distribution
15 activity, which involved the use of capacitors
16 and transformers.

17 Another is in that same unit. The
18 distribution of compressed air involved the use
19 of compressors which used PCB type fluids in them.

20 The second principal use was the
21 use of PCBs in the formulation of industrial
22 hydraulic fluids as part of the warehousing
23 function.

24 Q Okay. I don't understand what you mean by

1 industrial hydraulic fluids being used in a
2 warehouse function.

3 A The warehouse function which was under me, and
4 then in turn under a supervisor, one of its
5 activities involved the receiving of different
6 materials; the unloading of these materials in
7 the tanks, the pumping from those tanks into
8 other tanks of several ingredients and blending
9 according to some formula or formulation.

10 That blend would then be sold as
11 an article in commerce. And the blends that had
12 PCBs in them were industrial hydraulic fluids.

13 Q Okay. The first use that you mentioned was as a
14 user of electrical equipment and the distribution of
15 that equipment.

16 I assume that's at the Monsanto
17 Queeny plant you're talking about?

18 A Correct.

19 Q So, you would be more taking the things in and
20 distributing them somewhere in-house, rather than
21 shipping them out to customers.

22 Is that fair to say?

23 A I thought you were talking about distribution of
24 electrical energy.

1 Q Okay. I was talking about electrical equipment.

2 You were talking about energy?

3 A I'm talking about energy; not the equipment.

4 Q Okay.

5 A The equipment is in place and my workers would
6 operate and make sure the equipment is
7 distributing the electrical power that we
8 generated in the plant and we purchased from the
9 local utility company. It was distributed to
10 the plant properly and safely through the
11 transformers and capacitors installed by the
12 maintenance people.

13 Q All right. And I assume serviced by them, too?

14 A Yes.

15 Q Do you recall any particular precautions in that
16 time period, '61 through '64, regarding the
17 handling, use, repair or disposal of PCB
18 electrical equipment such as transformers?

19 A No.

20 Q Do you recall any labels, warning labels or
21 instruction labels being used on transformers in
22 that time period?

23 A No.

24 Q And then the second use is that of PCBs coming

1 in to you at Monsanto, at the Queeny plant in
2 warehousing, and your folks would take it and
3 blend it and turn it into whatever the finished
4 product was, I assume in drums or some such
5 thing, as an industrial hydraulic fluid?
6 A Correct.
7 Q Do you recall any material data sheets or
8 precautions or operating instructions in that
9 regard?
10 A Yes.
11 Q Of what type?
12 A They were part of the operating instructions
13 used by the employees to prepare these products.
14 And there was a section which referred to the
15 safety and proper handling of all chemicals that
16 they could have been exposed to.
17 Q Were there specific parts of that operating
18 instructions manual relating specifically to
19 PCBs?
20 A Yes.
21 Q And then there would be parts, I assume, relating
22 to other chemicals?
23 A Yes.
24 Q Do you recall any generalized types of warnings

1 or precautions in the operating manual in that
2 particular time period, '61 through '64?

3 A Under generalized topics, certainly those materials
4 that were flammable, there would be reference to
5 their flammability and the avoidance of flames and
6 sparks and the like.

7 For those materials that were
8 highly volatile and could cause fume problems, there
9 were warnings to have proper ventilation, avoid
10 prolonged breathing, and there
11 were references to the use of respiratory
12 protection as appropriate. It depends on the
13 material being handled.

14 Q Do you recall any generalized instructions or
15 safety precautions pertaining specifically to
16 PCBs in that time period?

17 A In a general way, yes. They referred to avoid
18 breathing of the fumes and avoid skin exposures;
19 skin contact.

20 Q Do you know if the operating instructions or
21 precautions at that time provided any sort of
22 safety equipment or safety devices to be used
23 around PCBs?

24 A Certainly, they did list references to gloves

1 and changes of clothing, respirators.

2 Q Do you have any idea -- you have knowledge of
3 those operating instructions in the '61 to '64
4 period. Do you have any idea how long they were
5 in place at Monsanto prior to that?

6 A This particular -- well, it's difficult to designate
7 exactly how long, because this particular unit
8 was assigned to this particular part of the plant
9 at the time I took over. So, it was a new unit
10 with new instructions, new safety sections and
11 the like.

12 The operations that were
13 consolidated in that unit had occurred at other
14 plants on a smaller scale, with identical kinds of
15 instructions regarding safety and handling.

16 The best that I can recall is
17 that similar instructions on safety and handling
18 probably date to the middle '50's, for these
19 industrial hydraulic fluids.

20 Q When there are such operations manuals or
21 operating instructions - I don't know exactly
22 what the right name is for that document - but
23 is it dated or numbered in some way so you know
24 when its effective date was?

1 A It's dated.

2 Q Do you recall ever seeing any of them concerning
3 PCBs dated back to the mid 1950's.

4 A I don't recall specifically, no.

5 Q Okay. We talked about what were some of the
6 problems with some of the earlier chemicals we
7 talked about.

8 What were some of the similar
9 problems with PCBs, if you recall?

10 A I would prefer to call them potential problems.

11 Q Okay.

12 A With PCBs, we were cautioned by our medical
13 experts to avoid breathing the fumes, because
14 they could result in the symptoms I described
15 earlier that resemble severe chest cold,
16 congestion, discomfort, pain in breathing, fluid
17 accumulation in the lungs.

18 We were also cautioned against
19 absorption through the skin, because the material
20 can cause an irritation of the skin, and it can be
21 quite severe depending on length of exposure
22 and amount.

23 And in extreme cases, there had
24 been some evidence that the workers exposed to

1 large amounts for long times were exhibiting a
2 skin condition referred to as chloracne.

3 Q Any other precautions that you can think of
4 relating to PCBs?

5 A No.

6 Q Do you recall any guidelines being provided as
7 to just how much of the chemical inhalation
8 would be a problem?

9 A Yes. I was made aware of a guideline issued by
10 a group of industrial hygienists that referred
11 to the amount of PCBs permissible in the working
12 environment.

13 As I recall, there were two types
14 of PCBs mentioned. They referred to them as
15 Monsanto's trademark Aroclor 1254 and 1242.

16 The Aroclor 1242 was permitted
17 at a little higher level. As I remember, it was
18 a milligram of the PCB for every cubic meter of air.

19 And for the Aroclor 1254, it was
20 a half a milligram per cubic meter of air.

21 Q The industrial hygienists you referred to, is
22 that AIHA that promulgated that particular
23 standard or suggested guideline?

24 A No. I believe this was the government

1 chemists -- government industrial hygienists,
2 ACGIH.

3 Q Do you recall reading anything actually
4 published in that time period by that group or
5 by somebody supporting their suggested
6 limitations?

7 A I recall both the ACGIH listing and a pamphlet
8 which I think was sponsored by the American
9 Institute of Industrial Hygienists.

10 Q And how about the absorption through the skin?
11 Any particular levels involved there or
12 suggested levels?

13 A I have never heard of a level.

14 Q Did the guidelines suggest that you should not
15 be exposed to a lot of it on your skin, or you
16 should not absorb a lot of it through your skin;
17 if either of those?

18 A It referred to exposure to undue amounts and
19 the recommendation of wearing protective gloves
20 or washing the soiled clothing and the like.

21 Q Okay. Was it suggested that you should not
22 have skin contact at all?

23 A I have never heard it expressed that way, no.

24 Q You never heard it expressed in, say, terms of

1 avoid skin contact?

2 A Yes. It was avoid skin contact, not don't have

3 any at all.

4 Q Okay. Avoid skin contact was their words?

5 A Yes.

6 Q Okay. Did your job duties and responsibility

7 change anytime in that '61 through '64

8 period as you've already described them to us?

9 A No.

10 Q Do you recall who you were reporting to in that

11 time period?

12 Who was the next step above you

13 as a general superintendent?

14 A The plant manager.

15 Q Do you recall who he was?

16 A Yes. Charles Smith.

17 Q Okay. And after 1964, I see here that you

18 became general superintendent of manufacturing,

19 but at a different plant.

20 A Yes.

21 Q What plant is W.G. Krummrich?

22 A That's a Monsanto plant located at Sauget,

23 Illinois, S-a-u-g-e-t.

24 Q I was tempted to call it Sauget. I guess that's

1 not right.

2 A Most of us are. It's a French family name.

3 Q Okay. And what did the Krummrich plant

4 manufacture?

5 A Oh, they manufactured several hundred different

6 chemicals.

7 Q Same as those at the Queeny plant?

8 A No. There were a few that were at both, but not

9 many.

10 Q Would it be part of a different division then, as

11 a plant goes, within Monsanto?

12 A No. It was the same division, a different group

13 of products.

14 Q And what division is that?

15 A At that time, it was called the Organic

16 Division.

17 Q Not specialty fluids yet?

18 A Those were a subgroup of the division.

19 Q A subgroup of the organic chemicals?

20 A Yes.

21 Q Okay. What did you do as a general

22 superintendent of manufacturing at Krummrich

23 plant?

24 A I was assigned about a fifth of the activities

1 at the Krummrich plant relating to
2 manufacturing.

3 Q I assume by that that there's about five general
4 superintendents then of manufacturing, and you're
5 one of the five?

6 A Yes.

7 Q And what area -- if you could, describe generally
8 the nature of the chemicals that you were in
9 charge of manufacturing in that period.

10 A I had reporting to me the operation that
11 produced phenol.

12 Q Phenol?

13 A Phenol, P-h-e-n-o-l; chlorinated Phenol; one of
14 the rubber chemicals.

15 My memory fails me. I can't
16 recall the other units at the moment.

17 Q Was PCBs part of your responsibility in that
18 time period?

19 A No.

20 Q What group was it that was in charge of PCBs,
21 assuming there were PCBs at that particular plant?

22 MR. SCOTT: That's two questions, so
23 why don't you do them one at a time.

24 MR. LEDBETTER: Okay. Let me go to the

1 first one.

2 BY MR. LEDBETTER:

3 Q Were PCBs manufactured at the Krummrich plant?

4 A Yes.

5 Q And what group was it that was responsible for

6 those?

7 A It doesn't have a name. It reported to one

8 of the general superintendents of manufacturing.

9 Q Do you recall who that fellow was?

10 A Paul Heisler, H-e-i-s-l-e-r.

11 Q And he was a general superintendent like

12 yourself of manufacturing?

13 A Correct.

14 Q In that '64 to '65 period, did you have any

15 involvement with PCBs, although not necessarily

16 directly under your authority?

17 A No.

18 Q Do you recall any specific regulations or

19 operating instructions pertaining to the

20 manufacture, handling and safety regarding PCBs

21 at the Krummrich plant?

22 A No.

23 Q Okay. And then it shows here in 1965, you became

24 plant manager at the Anniston plant.

1 A Yes.

2 Q That was in Alabama, right?

3 A Yes.

4 Q And what was manufactured at the Anniston plant?

5 A PCBs; biphenyl; hydrogenated biphenyl; PCTs,
6 polychlorinated terphenyls; paranitro Phenol;
7 chlorine; muriatic acid; sodium Hydroxide or a
8 caustic soda, same thing; and a chemical
9 pesticide or insecticide, methylparathion and
10 ethylparathion.

11 Q What were your job duties and responsibilities
12 as the plant manager of the Anniston plant?

13 A I suppose you could describe it as I was
14 responsible for everything and anything at the
15 plant.

16 Q So, you'd have people reporting directly to you,
17 including the superintendents that we talked
18 about earlier?

19 A Yes.

20 Q Was there a division level between yourself and
21 the superintendents or a department level?

22 A No.

23 Q There wasn't like an assistant plant manager or
24 something of that sort?

1 A No.

2 Q Okay. So, the superintendents over all of the
3 areas including manufacturing, would report
4 directly to you as the plant manager?

5 A Yes.

6 Q Who did you report to at Monsanto, if any
7 particular person?

8 A I reported to the Director of Manufacturing of
9 the Organic Chemicals Division.

10 Q Do you recall who that was in that time period,
11 '65 through '70?

12 A There were two of them. Initially, it was
13 Robert Soden, S-o-d-e-n. And he was replaced by
14 Raymond Stratmeyer, S-t-r-a-t-m-e-y-e-r.

15 Q At the time that you were in the Anniston plant
16 in that five-year period, '65 through '70, do you
17 recall any specific involvement you had with
18 PCBs?

19 A Well, I was responsible for the manufacturing
20 unit.

21 I don't know that that involved --
22 was any different than the involvement with all
23 the other units in the plant.

24 Q Okay. On some sort of a comparison scale if

1 it's possible, is there any particular one
2 product that was manufactured more than any
3 other product at the Anniston plant?

4 MR. SCOTT: Do you mean in terms
5 of volume turned out?

6 MR. LEDBETTER: Yes. Volume
7 turned out; total production.

8 THE WITNESS: I, of course, don't
9 recall the exact numbers, but as best I recall,
10 the insecticide volume was about equal to the
11 PCB volume.

12 BY MR. LEDBETTER:

13 Q Were those the two largest you believe at that
14 time period?

15 A Yes.

16 Q Do you recall any specific plant directives or
17 guidelines relating to the handling or safety in
18 the utilization of PCBs or the manufacture of
19 PCBs?

20 A Yes.

21 Q What were those?

22 A These were the documents I referred to earlier,
23 the Standard Manufacturing Process Manual and
24 the Standard Operating Instructions.

1 Q Is that the correct title, how we can refer to
2 it; Standard Operating Instructions?

3 A It varied slightly from time to time and from
4 plant to plant.

5 If it isn't Standard Operating
6 Instructions, it might be referred to as
7 Standard Manufacturing Procedures.

8 Q But they would be very similar documents, to your
9 understanding; just with different names?

10 A Correct. It's colloquially known as either SOPs or
11 SMPs.

12 Q You told us a little bit about the Standard
13 Manufacturing Process Manual. That's the
14 technical literature on the manufacture of given
15 chemicals, correct?

16 A When the word "Process" appears on the title,
17 that is, in essence, the Bible of the process;
18 all the technical data and anything needed to
19 know about the process is in that document.

20 Q Are there precautionary statements in terms of
21 handling or exposure or potential exposure in that
22 manual?

23 A Yes.

24 Q To the various chemicals?

55

1 A Yes.

2 Q Okay. Would those be mocked pretty much in the
3 Standard Operating Instructions or Standard
4 Manufacturing Procedures; similar wording,
5 similar instructions?

6 A Yes.

7 Q Do you recall if there's one specific element of
8 PCB that made it particularly toxic or would
9 warrant some specific instructions regarding its
10 handling?

11 A I would not call any one element. I would
12 suggest that the message involved the two areas
13 we talked about earlier. This is effect on the
14 respiratory system and the effect on skin.

15 Q Say, for example, that we were just -- chlorine
16 first is a part of polychlorinated biphenyl, right?
17 Chlorine is part of that formula?

18 A Yes.

19 Q Let's say there were just the chlorine.
20 Would that have an effect on the
21 respiratory system?

22 A Oh, yes.

23 Q And how about the skin?

24 A It would affect the skin also.

1 Q If we left out the chlorine and we just had
2 biphenyl or diphenyl, would either or both of
3 those by themselves affect the respiratory
4 system and the skin?
5 A I'm confused by leaving out chlorine.
6 I didn't mean to imply earlier --
7 in my references to chlorine, I was thinking of
8 chlorine as the gas itself before it's reacted to
9 make PCBs.
10 Does that still fit your question?
11 Q Okay. I think it still fits the question.
12 Let's say that we haven't gotten
13 to the point yet where the chlorine gas is added
14 to the chemical.
15 A Okay.
16 Q What product do we have at that point, prior to
17 chlorine being added?
18 A Biphenyl.
19 Q Is biphenyl, prior to the chlorine being added,
20 a potential problem to the respiratory system?
21 A Yes.
22 Q By itself?
23 A Yes.
24 Q The fumes would cause particular respiratory

1 problems if inhaled?

2 A Yes.

3 Q And how about biphenyl on contact with the skin?

4 Would that create a problem?

5 A No; unless it's a thermal problem.

6 Q Okay. You mentioned that earlier. If it's

7 hot, then it can certainly burn you?

8 A Yes.

9 Q Other than that, skin contact, to your

10 knowledge, wouldn't be particularly toxic?

11 A No.

12 Q Would the addition of chlorine make the biphenyl

13 more toxic to the respiratory system?

14 A It's my understanding it would, yes.

15 Q Same question as to adding chlorine to biphenyl,

16 and its effect to the skin; dermal effect?

17 A Yes.

18 Q Okay. Perhaps we can back up a little bit.

19 PCB, as I understand it, stands

20 for polychlorinated biphenyl; is that correct?

21 A Biphenyl.

22 Q Biphenyl. How does that material come to be?

23 What does it start from?

24 A How far back should we go?

1 Q Well, let's not go back in nature.

2 Let's say we start with a basic
3 raw material that's going to be used to
4 ultimately manufacture PCB.

5 What's that basic raw material?

6 A The basic raw materials is biphenyl and chlorine.

7 Q How do you make biphenyl?

8 A The procedure that I'm familiar with starts with
9 benzene, at high temperatures and pressure, to
10 make the biphenyl.

11 Q Okay. What is benzene?

12 A That's a material that's obtained out of crude
13 oil, petroleum fraction, or it can also be
14 obtained out of cold tar distillation. And it
15 consists of carbon and hydrogen. And the
16 chemist graphically shows this when he
17 communicates, as a six-sided hexagon-shaped
18 diagram. Each of the corners of the hexagon
19 represents the location of a carbon. The carbons
20 are associated with each other, and on each
21 carbon, there's a hydrogen.

22 The combination at that
23 configuration is known as benzene.

24 Q Okay. And then how do we get from benzene to

1 diphenyl again?

2 A The two benzenes join each other at a
3 point-to-point; carbon-to carbon. So, we now have
4 two benzene rings or hexagons combining to make a
5 double hexagon or a double biphenyl; double
6 phenyl.

7 Q And hence the "bi"?

8 A Correct.

9 Q Okay. Is that a heating process that it takes
10 to get to that point; to the biphenyl?

11 A Yes.

12 Q Do you recall what kind of temperatures are involved,
13 roughly?

14 A Very roughly in the -- about 400 degrees
15 centigrade. I recall the vessels glowing at
16 night. They were red hot.

17 So, it's high.

18 Q When you start with benzene only, as far as its
19 toxic effect, would it be a respiratory problem;
20 benzene alone?

21 A It can be, but it takes fairly high volumes to
22 irritate the respiratory system.

23 Q It becomes more of an irritant to the
24 respiratory system after you've heated it and

60

1 turned it into biphenyl?

2 A Yes.

3 Q Once it is biphenyl, how do we get it from the
4 biphenyl condition to the chlorinated biphenyl
5 position?

6 A The biphenyl at room temperature is a solid. It
7 looks very much like white candle wax. So it must
8 be heated before it can be pumped. It's
9 transferred into a tank, and in this tank there
10 is normally a device for introducing chlorine gas.
11 It's called sparging.

12 And in the presence of a catalyst
13 to make the reaction go faster and more
14 efficiently - and this catalyst in this case is iron
15 chloride - chlorine is introduced. It bubbles
16 through the molten biphenyl, the chlorine
17 randomly attaches itself to carbons that are
18 present on the double phenyl group, displacing
19 hydrogen. That displaced hydrogen combines with
20 other chlorine to form hydrochloric gas.

21 So, we have forming in that
22 vessel, roughly three different chemical
23 systems. One is the hydrochloric gas, the other
24 is the PCBs, and the third is the tars, the

1 miscellaneous unwanted residue.

2 Q Okay. Would the gas be appropriately called a
3 byproduct or a residue?

4 A They

5 Q It kind of depends on what you're trying to
6 make, doesn't it, as to which one you'd call
7 them?

8 A True.

9 Q So, if you're trying to make PCBs, the other two
10 could properly be called a byproduct or residue?

11 A Correct.

12 Q We have now from this manufacturing process that
13 you've described, we've identified three things
14 that come from it; one is PCBs. But by the name
15 that we've used so far, we've got chlorinated
16 biphenyl.

17 How do we come up with poly?

18 A The expression "poly" refers to the fact that
19 there's more than one chlorine associated with
20 each biphenyl.

21 Q How do you tell the chlorines, how many of them
22 are going to go to each biphenyl?

23 A I don't understand the question.

24 Q You told me that chlorine is bubbled through

1 molten biphenyl and you replace the hydrogen
2 with a chlorine.
3 A That's correct.
4 Q It's at random?
5 A Correct.
6 Q When it attaches, the hydrogen is displaced and,
7 hopefully, you end up with more than one
8 chlorine on each one; you would have then
9 polychlorinated, correct?
10 A Correct.
11 Q If you only ended up with one, it wouldn't be
12 poly?
13 A Correct.
14 Q How do you control how many chlorine molecules
15 attach?
16 A The industrial PCB that is marketed is a mixture
17 of the 1, 2, 3, 4, all the way up to
18 10, possible 10 chlorines attached to the
19 biphenyl.

20 In the manufacturing process, we
21 did not control in any way by counting, if you
22 will, the number of chlorines that are attached.
23 By experience, we knew that after a given period
24 of time, we could expect a certain mixture to be

1 created. And we would measure the completeness of
2 that reaction by taking a sample and determining
3 the specific gravity of that liquid.

4 In other words, how heavy a
5 liquid is it with respect to water as the
6 standard.

7 Q And you have some sort of comparison scale that
8 you evaluate the specific gravity of that
9 product? You expect it to be whatever weight
10 compared to water?

11 A Yes.

12 Q And if you match that weight, then you know what
13 product you have?

14 A Yes. The operator would know that he's
15 finished; to turn off the chlorine.

16 Q What happens if he goes further?

17 A He is forced, then, to go into the next type of
18 commercial product; the higher chlorine
19 material.

20 Q Okay. Again, you said that this next step from
21 biphenyl requires bringing the biphenyl to a
22 molten condition.

23 Do you know what kind of heat is
24 involved there, approximately?

1 A Approximately -- it's over 100 degrees centigrade.
2 About 150 to 200 degrees centigrade.

3 Q Okay. I'm still a little bit off to determine
4 through what process we stop at, say, an
5 Aroclor 42 percent chlorine and an Aroclor
6 that's 54 or an Aroclor that's 60.

7 Is that a matter of time and
8 temperature?

9 A The temperatures are fixed. It then becomes a
10 matter of time, confirmed by this sampling I
11 mentioned. And when the operator reaches a given
12 specific gravity, he knows that he now has a
13 mixture in his reaction vessel that could give
14 him the product that he set out to make, whether
15 it's Aroclor 1242, or Aroclor 1254 or 1260.

16 At that point, as I indicated, he
17 turns off the chlorine, and he's now ready to
18 proceed to refining this mixture to end up with
19 the product that he set out to make.

20 Q What further refinement has to be done?

21 A There are several steps involved.

22 Initially, air is bubbled through
23 this reaction mass to remove the gas that is
24 still trapped. And by gas, I'm talking about

1 the unreacted chlorine and the hydrogen chloride
2 that was formed.

3 The next step is to add something
4 to neutralize the hydrogen chloride that the air
5 wasn't able to displace or sweep out. Monsanto
6 used lime to neutralize.

7 Then the mass was heated to
8 temperatures somewhere in the 300 degrees
9 centigrade range with a slight vacuum pulled on
10 the tank to suck off the vapors.

11 The vapors are drawn up overhead
12 and passed through an air cooler, very much like
13 a car radiator. It's a large, industrial size
14 unit. The vapors are condensed, they're
15 collected in a tank, and that becomes the
16 finished product.

17 When the vapor stops coming off
18 the reaction vessel, the process is finished. And
19 the material still remaining in the pot is the
20 residue or the tarry material that I referred to
21 earlier.

22 Q Okay. Is there any particular guideline to how
23 much of the -- let me strike that and start over
24 again.

1 In the finished product, is there
2 a test done to see if it's a pure, finished
3 product or if it still has some byproduct in it?

4 A The material that is distilled and collected is
5 agitated to get uniformity. A sample is taken
6 and is sent to the control laboratory where the
7 chemist then determines if it meets the
8 specifications for that particular PCB mixture.

9 Q Were there any tolerances provided that you're
10 aware of?

11 A Yes, there were, as I recall, a full page of
12 specifications that it must meet, and there are
13 ranges.

14 Q The chemical properties -- the lines of the
15 chemical, so to speak, on a graph-like thing? Is
16 that the idea?

17 A Well, at that point in time, no.

18 It was primarily a matter of the
19 amount of chlorine, its specific gravity, its
20 color, its dielectric consonant, vapor pressure.
21 I've forgotten all of the features that it must
22 meet.

23 So, this was a combination of
24 chemical and physical properties.

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1 Q Now, the finished product that we're talking
2 about at this particular time, is PCBs in a
3 specific Aroclor?

4 A Correct.

5 Q So, there can be a lot more different Aroclors,
6 all indicating a different chlorine level?

7 A Correct.

8 Q Do you know if there's any other impurities or
9 byproducts in that, even though you haven't
10 analyzed it to see if it's a certain Aroclor?

11 Is there any way to tell what kind
12 of impurities or byproducts are left in
13 there?

14 A Not at that time.

15 Q Okay. There was no specific test done to
16 determine if there were some byproducts in that
17 final Aroclor product?

18 A Correct.

19 Q From your experience as a chemist, would you
20 expect that there would be certain byproducts in
21 the finished product?

22 A Yes.

23 Q And what would those be?

24 A I don't know.

1 Q Would you expect that dibenzo Furans may be part
2 of the byproduct?

3 A Possibly.

4 Q No others that you can recollect?

5 MR. SCOTT: He said he doesn't know.

6 MR. LEDBETTER: He's not sure or
7 he doesn't know.

8 MR. SCOTT: The answer was: I
9 don't know.

10 Let's not change his answer.

11 BY MR. LEDBETTER:

12 Q Do you recall any analysis done on Aroclor
13 products to determine the existence of
14 byproducts?

15 A At what point in time?

16 Q After the finished product is distilled through
17 condensation, and you've got a finished product.
18 Any time along, in your experience, that an
19 analysis was done to determine the existence of
20 byproducts?

21 MR. SCOTT: You mean as a part
22 of the manufacturing process?

23 MR. LEDBETTER: Yes.

24 THE WITNESS: What year?

1 BY MR. LEDBETTER:

2 Q '65 through 1970.

3 A No.

4 Q Do you recall there being something done
5 thereafter?

6 A Yes.

7 Q What was that?

8 A There were some analytical research studies
9 made - not production and control studies
10 analyses - to determine the presence of the
11 chloro dibenzo Furans in these products.

12 Q Do you recall the results of those analyses?

13 A The initial results came back to me as non-
14 detectible.

15 Q And later?

16 A I was not involved when further studies were
17 made.

18 But I understand further studies
19 were made.

20 Q Do you recall what the results of those were
21 through the information that you received?

22 A I received information that they found low
23 levels using more sophisticated methodologies.

24 Q Just so that we have the terms straight, what is

1 the content of, say, Aroclor 1260?

2 What is that made up of?

3 A It's made up of a mixture of biphenyls that
4 contain chlorine. And it's predominantly in the
5 five chlorine range, but it does contain two
6 chlorines, three, four, six, some seven chlorine
7 types of PCBs.

8 Altogether, the amount of
9 chlorine present is calculated to be 60 percent
10 by weight of the total weight.

11 Q And then what is the rest? Diphenyl?

12 A It's carbon and hydrogen.

13 Q Do you recall roughly the percentages of each?

14 A No, I don't.

15 Q But the carbon and hydrogen would make up the
16 remaining 40 percent?

17 A Yes.

18 Q Okay. And then would it be a correct assumption
19 if we were talking Aroclor 1254, that it would
20 be 54 percent chlorine and the remainder of 46
21 percent being hydrogen and carbon?

22 A Correct.

23 Q Do you recall of the Aroclors, how many of the
24 1200 series Aroclors there were?

1 A Yes. I think I can run down the list.

2 Q What were those?

3 A 1221, 1232, 1242, 1248, 1254, 1260, and 1268.

4 Q Now, the only difference in those - if I can say
5 the only difference - is the amount of chlorine
6 in each; is that correct?

7 A That's correct.

8 Well, that's a simple way to
9 describe it. They are more complex in terms of
10 the actual chemistry that's present in each
11 mixture.

12 Q Okay. Meaning the number of chlorine molecules
13 attaching to each?

14 Is that fair to say?

15 A It's the number and the position. It's not only
16 three chlorines, but where are those three.

17 There are many combinations
18 possible, so they can be complex mixtures.

19 Q Okay. Does it matter, as far as the eventual
20 product being completed?

21 Say, if it's desired to have
22 Aroclor 1260, does it matter where the chlorine
23 molecules attach and how many?

24 A I don't know.

1 Q It's never been studied, to your knowledge?

2 A It could have been studied, but I have not.

3 It is possible to have 60 percent
4 by weight of chlorine and still have the wrong
5 properties.

6 Q Do you recall if there were other Aroclors
7 other than the 1200 series?

8 A Yes.

9 Q What were those?

10 A These were polychlorinated terphenyls.

11 And there was at least two that I
12 recall that were blends of PCBs and PCTs.

13 Q Was there any particular Aroclors that were
14 used primarily in electrical equipment such as
15 transformers and capacitors?

16 A Yes.

17 Q What were those?

18 A The Aroclor 1221, Aroclor 1242, Aroclor 1248,
19 Aroclor 1254, Aroclor 1260.

20 Q In the '65 to '70 period, do you recall what the
21 major use of Aroclors were, if there was a
22 specific category for the majority use?

23 A To my recollection, it was the dielectric
24 application.

1 Q Was there any particular criteria used to
2 determine which type of Aroclor would be placed
3 in which type of electrical equipment?
4 A There were criteria, but they were established
5 by the customer.
6 Q Okay. Whoever was buying the product would
7 require a certain Aroclor to be provided by
8 Monsanto?
9 A Correct.
10 Q Do you remember --
11 My understanding is PCB is
12 synonymous with Aroclor. Is that fair to say?
13 A No.
14 Q Is PCB synonymous with the Aroclor 1200 series?
15 A Yes.
16 Q So, if we move out of that group, where we get
17 to the polychlorinated terphenyls, for example,
18 then we don't have PCBs anymore?
19 A That is correct.
20 Q It's still the same properties, as far as the
21 adverse or potential adverse reactions to PCBs
22 and PCTs?
23 A No.
24 Q How does it change?

1 We've already talked about PCBs,
2 so just talk about PCTs, if you would.

3 A The PCTs were solid materials, so the problems
4 or the potential problems with fumes were
5 decreased considerably.

6 If misused, dusting problems
7 could occur, although they did not form dust
8 easily. So, the respiratory caution there was
9 don't breathe dust, as distinguished from don't
10 breathe the fumes.

11 The effect of the dust was similar
12 to other dusty materials in forming irritation,
13 as distinguished from a chemical -- a corrosive-type
14 of effect on the respiratory system.

15 The effect on the skin was not as
16 pronounced or even considered worthy of any
17 special attention. It just did not give the
18 reddening of the skin or the chloracne that the
19 PCBs were known to have the potential for
20 causing.

21 Q Okay. Are you familiar with the term "Pyranol"?

22 A Yes.

23 Q Does Pyranol suggest a specific Aroclor?

24 A No.

1 Q Pyranol could be a number of different
2 Aroclors?

3 A Yes.

4 Q Is Pyranol containing something more than
5 Aroclor, whatever the blend is?

6 A It's possible.

7 Q Do you have any personal knowledge as to what is
8 contained in Pyranol?

9 MR. DINON: Objection;
10 foundation as to time.

11 BY MR. LEDBETTER:

12 Q '65 to '70.

13 A Some Pyranols contained chloro benzenes;
14 trichloro or tetrachloro or a blend of the two.

15 Q And they also contained an Aroclor blend?

16 A Yes.

17 Q Do you know how much of which?

18 A I don't recall the recipes.

19 Q Okay. Do you know from early experience or
20 other knowledge attained somehow, what might have
21 been in Pyranols back in the '40's or '50's?

22 A My understanding is that the Pyranols early on
23 were still blends of PCBs with chloro benzenes.
24 Those that were blends.

1 Q Are you implying that there were some that were
2 not blends?

3 A Yes.

4 Q And would that mean some are straight PCBs or
5 straight chloro benzenes or bichloro benzenes?

6 A Some Pyranols were straight PCBs.

7 Q And "Pyranol," that's the GE trade name?

8 A Yes.

9 Q Do you know what type of Pyranol was provided to
10 GE in 1941?

11 A No.

12 MR. SCOTT: What type of Pyranol
13 was provided to GE? That's GE's product, isn't it?

14 MR. LEDBETTER: It's GE's trade
15 name.

16 MR. SCOTT: Well, I understand
17 that.

18 Never mind. Go ahead.

19 MR. LEDBETTER: Well, perhaps
20 I can back up and make sure that I understand.

21 BY MR. LEDBETTER:

22 Q When a customer asks for, say, a
23 certain chemical to be provided by Monsanto, and
24 say it's Aroclor, if it's to be used in a

1 transformer application, who is it that blends
2 the chemical to be finally put into the piece of
3 electrical equipment, if it's to be used for
4 that purpose?

5 MR. DINON: Objection;
6 foundation. It's vague and overbroad.

7 What time frame are you referring
8 to? What customer, what application?

9 BY MR. LEDBETTER:

10 Q Go ahead and answer over all of those
11 objections, if you can.

12 A My answer to your question is: Monsanto can do
13 the blending and packaging and shipping or the
14 customer may do his own blending.

15 Q If it's a customer that does its own
16 blending, might I assume by that that the
17 customer would just ask you to send a certain
18 amount of Aroclor product to them?

19 MR. DINON: Same objection.

20 THE WITNESS: Yes.

21 BY MR. LEDBETTER:

22 Q Do you recall, in your experience with GE, how
23 it was done for their Pyranols?

24 MR. DINON: Same objection.

1 THE WITNESS: Both ways.

2 BY MR. LEDBETTER:

3 Q Do you recall what the term "air blowing" means?

4 Is that synonymous with the air

5 bubbling purification system that you indicated

6 earlier?

7 A To me, it would be, yes.

8 Q Air blowing?

9 A Yes.

10 Q Is that a term of art?

11 A Yes.

12 Q Okay. How about HCL? Is that hydrochlorine

13 or hydrochloride?

14 A HCL is the chemical acronym for hydrogen chloride

15 gas, which when dissolved in water, makes

16 hydrochloric acid, commonly known as muriatic

17 acid.

18 Q Now, the Aroclor blend that is the final

19 product, it no longer contains the hydrochloride

20 gas, right?

21 A That is correct.

22 Q With reheating, would hydrochloride gas be

23 created?

24 A At certain critical temperatures, the PCB can

1 degrade, forming hydrogen chloride gas.

2 Q Is that a method that's used to incinerate PCB

3 now, for example?

4 A When PCBs are incinerated, HCL is formed, yes.

5 Q Any other byproduct, or any other final product?

6 A Under the ultimate conditions, the final

7 products include not only the HCL, but H2O, which

8 is water, and CO2, carbon dioxide.

9 Q Any other byproduct left after that?

10 A No.

11 Q In the 1965 to 1970 period, do you recall

12 reviewing any specific studies or tests relating

13 to the potential toxicity of PCBs?

14 A Yes.

15 Q Do you recall any of those in detail or by

16 identification?

17 A I recall discussions with the plant medical

18 doctor, as well as Monsanto's Corporate Medical

19 Director.

20 The final understanding I had

21 from these discussions - and they occurred on

22 more than one occasion - was the cautioning by

23 both medical professionals to avoid breathing

24 and skin contact.

80

1 And these were based on some
2 studies that I understood had taken place in the
3 '30's, and there were some animal studies repeated
4 in the '50s and '60s, which confirmed that PCBs
5 can cause skin irritation, skin damage. As I
6 understood it, they were able to create a
7 chloracne on some rabbits that were tested; the
8 skin on rabbits that were tested.

9 I was also led to believe that when
10 the PCBs were first manufactured by Swann Chemical
11 Company, the predecessor company to Monsanto's
12 Anniston, Alabama plant, that the operators
13 experienced evidence of chloracne under the
14 conditions that existed at that time, which were
15 difficult to determine exactly. But the belief
16 was that there was undue exposure.

17 Q Now, the tests that you're referring to done at
18 what was then Swann Chemical Company that was done
19 in the '30s?

20 A Yes. There were tests requested by Swann
21 Chemical which I recall were completed for Swann
22 Chemical before Monsanto purchased Swann
23 Chemical, which was in the -- all this happened
24 in the '30s; pretty close to the time that

1 Monsanto purchased Swann Chemical Company.

2 Q Okay. As I understand it, that purchase took
3 place in 1935; is that correct?

4 A Yes.

5 Q Swann had some studies done or at least conducted
6 on their behalf sometime prior to 1935?

7 A Correct.

8 Q Do you recall any particular names or title of
9 any of those studies?

10 A No, I don't.

11 MR. LEDBETTER: Let's mark this as
12 Number 1.

13 (Whereupon Deposition Exhibit
14 Number 1 marked by reporter)

15 BY MR. LEDBETTER:

16 Q Mr. Papageorge, let me show you a study that was
17 conducted, I believe in 1936, entitled An
18 Acneform Dermatargosis by Jack Jones and
19 Herbert Alden, and I'll ask if you can identify or
20 if you've seen that study before?

21 MR. SCOTT: I gather you represent
22 that the handwritten margin inter alia and the
23 underlining are not part of the study?

24 MR. LEDBETTER: I would

1 acknowledge that, yes.

2 MR. SCOTT: Okay.

3 THE WITNESS: I have never seen
4 this document before.

5 BY MR. LEDBETTER:

6 Q Sir, was Monsanto involved in the manufacture of
7 PCBs anywhere prior to 1935?

8 A No.

9 Q Are you aware of any companies that manufactured
10 PCBs other than Swann Chemical, prior to
11 1935?

12 A No.

13 Q So, if it's a PCB operation prior to 1935, would
14 it be a safe assumption that it's Swann Chemical?

15 A No, because I don't know who else might have been
16 making PCBs at the time.

17 Q Okay. Sir, are you aware of a study done by
18 Louis Schwartz on dermatitis from synthetic
19 resins and waxes?

20 A I was aware of some studies involving waxes. I don't
21 recall the names of the authors of that report.

22 (Whereupon Deposition Exhibit
23 Number 2 marked by reporter)

24

1 BY MR. LEDBETTER:

2 Q Let me show you what's been marked as Exhibit
3 Number 2, which is a study conducted by one
4 Louis Schwartz, labeled, I believe, in 1936, and
5 ask if you've seen that particular study?

6 A No, I have not.

7 Q Okay. Again, it's not one that you're familiar
8 with in your readings?

9 A That is correct.

10 MR. SCOTT: Counsel, can you
11 decipher the writing in the upper right-hand
12 corner, which apparently refers to the journal
13 where this was taken from?

14 MR. LEDBETTER: Archives of
15 Dermatological and --

16 MR. SCOTT: Symposium Archives?

17 MR. LEDBETTER: Archives of
18 Dermatologic, and something like that.

19 MR. DECKER: What's the date of
20 that report, J.D.?

21 MR. LEDBETTER: 1936.

22 MR. SCOTT: Reported at Volume 33,
23 page 1022, Archives of whatever it is.

24

1 BY MR. LEDBETTER:

2 Q Sir, in the time that you were at Monsanto, do
3 you recall having any particular source of studies
4 and such things, independent studies, done by persons
5 evaluating the potential toxic effects of PCBs?

6 A Yes.

7 (Whereupon brief pause in proceedings)

8 BY MR. LEDBETTER:

9 Q The last question I sought was whether in your
10 time at Monsanto, you had reviewed certain studies
11 and analyses of PCB or potential toxic effects of
12 PCB. And you said that you had done that sort of
13 thing somewhere at Monsanto, correct?

14 A Yes.

15 Q And where were those documents made available to
16 you, or how were they made available?

17 A They were available in Monsanto's corporate
18 medical files. Copies of some of them were made
19 for my personal use. The remaining documents
20 were available to me at the Medical Director's office,
21 where I could peruse them whenever I wished to.

22 Q And you did have the opportunity to use all three of
23 those sources that you've identified to see the
24 documents?

1 A Yes.

2 Q Where were the corporate medical files kept at
3 the time that you were aware of them?

4 A In Monsanto's general headquarters in St. Louis.

5 Q Any particular division, subdivision or office;
6 anything of that sort?

7 A The office of the Corporate Medical Director.

8 Q You mentioned back in '65 to '70, that studies
9 or tests had been made available to you through
10 the plant medical doctor and also through the
11 Corporate Medical Director, correct?

12 MR SCOTT: No. I think you're
13 mischaracterizing his testimony.

14 What he said was that he had had
15 discussions with the Corporate Medical Director
16 and the plant doctor.

17 I don't think he referenced
18 studies that he saw between '65 and '70. I may
19 be wrong.

20 MR. LEDBETTER: Okay. Well, let's
21 let him answer that question.

22 BY MR. LEDBETTER:

23 Q Did you see studies, or did you discuss those
24 studies, in that '65 to '70 period, with the plant

1 medical doctor and the Monsanto Corporate Medical
2 Director?

3 A I did not see any reports. I was informed of
4 these studies during the discussions with the two
5 medical professionals.

6 Q All right. And who was the plant medical doctor
7 at that time, '65 through '70?

8 MR. SCOTT: You're talking about
9 the Anniston plant?

10 MR. LEDBETTER: Yes. That's
11 right.

12 THE WITNESS: Dr. James Francis.

13 BY MR. LEDBETTER:

14 Q Was he a full-time plant medical doctor?

15 A No.

16 Q Was he an M.D.?

17 A Yes.

18 Q How often was he at the plant; the Anniston
19 plant?

20 A Monday through Friday, daily, for two to three
21 hours every morning, and was available by telephone
22 for emergencies.

23 Q And then who was the Monsanto Corporate Medical
24 Director during that period of time?

1 A Dr. R. Emmet Kelly.

2 Q Okay. Do you know who replaced Dr. Kelly as
3 plant Medical Director?

4 A Dr. Kelly was not the plant Medical Director.

5 Q I'm sorry. The Corporate Medical Director.

6 A Dr. George Roush, R-o-u-s-h.

7 Q Is Mr. Roush still the Corporate Medical
8 Director?

9 MR. SCOTT: Dr. Roush.

10 THE WITNESS: Dr. Roush is still
11 today the Corporate Medical Director.

12 BY MR. LEDBETTER:

13 Q Okay. So, it was with Dr. Francis
14 and Dr. Kelly that you had these discussions
15 regarding the studies and tests regarding the
16 potential toxicity of PCB?

17 A Yes.

18 Q Do you recall what you were told at that time?

19 A Yes.

20 Q And what was that?

21 A That too much exposure to fumes can cause respiratory
22 problems; excessive exposure on the skin can
23 cause severe skin damage; or it can eventually
24 lead to chloracne, indicating high exposures over

1 long periods of time.

2 Q Did anybody ever attempt to put in a dosage that
3 would be too much exposure via inhalation?

4 A The reference was made in those earlier documents
5 we described put out by the American Congress of
6 Governmental Industrial Hygienists and the
7 publication put out by the American Institute of
8 Industrial Hygienists.

9 Q That was the one milligram per cubic meter and
10 the one and-a-half milligram per cubic meter?

11 A Yes.

12 Q That was for 1242 and 1254, correct?

13 A Yes.

14 Q Do you recall any specific guidelines for
15 Aroclor 1260?

16 A I was told to treat Aroclor 1260 the way we
17 treated Aroclor 1254.

18 Q I believe earlier you indicated that there was a
19 relationship in potential toxicity as to
20 inhalation and skin contact as chlorine was
21 added, correct?

22 A Yes.

23 Q Would it be a fair assumption that at least
24 marginally, the potential toxicity would be

1 greater with 1260 than with 1254?

2 A I don't know.

3 Q But you do know that the potential toxicity, at
4 least in terms of the suggested inhalation
5 amounts, was more strict for 1254 than it was for
6 1242?

7 A Yes.

8 Q Do you recall when you first read a study
9 regarding potential toxicity of PCBs?

10 A Early 1970.

11 Q And do you recall what that was; the first series
12 that you read at that time?

13 A I don't recall the title, but I believe I recall
14 an investigator, Dr. Drinker. Dr. Drinker is the
15 name I recall.

16 And I also recall a reference to
17 coatings on electrical cables. I recall the
18 reference to both chlorinated naphthalenes and
19 chlorinated biphenyls.

20 MR. LEDBETTER: Would you mark
21 that as 3, please, and this one Number 4.

22 (Whereupon Deposition Exhibit
23 Numbers 3 & 4 marked by reporter)

24

1 BY MR. LEDBETTER:

2 Q I show you now what has been marked as Deposition
3 Exhibit Number 3 and Number 4. And first, as to
4 Number 3, I'll ask if you can identify that
5 particular study?

6 A This appears to be a copy of a publication from
7 the Journal of Industrial Hygiene and Toxicology,
8 entitled The Problem of Possible Systemic Effect
9 From Certain Chlorinated Hydrocarbons. And the
10 authors are Drs. Drinker, Warren and Bennett.

11 Q Is that by chance one of the studies that you
12 recall reading sometime in 1970?

13 A It appears to be; yes, sir.

14 Q Okay. And then I'll ask the same thing as to
15 Deposition Exhibit Number 4, and ask if you can
16 identify that particular document?

17 A It's a copy of an article that appeared in the Journal
18 of Industrial Hygiene and Toxicology in February,
19 1938, Morphological Changes in the Livers of Rats
20 Resulting from Exposure to Certain Chlorinated
21 Hydrocarbons.

22 Q Is that again one of those that you recall from
23 1970, that reading in 1970?

24 A I don't recall this rat study, no.

1 Q Okay. Do you recall reading that one since?

2 A Not clearly, no.

3 Q Okay.

4 (Whereupon Deposition Exhibit
5 Number 5 marked by reporter)

6 BY MR. LEDBETTER:

7 Q Sir, I show you another one that has been marked
8 as Deposition Exhibit Number 5, from the American
9 Journal of Hygiene, entitled Liver Lesions Caused
10 by Chlorinated Naphthalene, and ask if that may be
11 one of those that you were referring to earlier?

12 A I do not recall this document.

13 Q Okay.

14 MR. SCOTT: Counsel, are you going
15 to give these to our distinguished court reporter
16 so she can copy them and send them to us?

17 MR. LEDBETTER: Yes, I will do
18 that; of the ones he can identify.

19 MR. SCOTT: The ones that are
20 marked as exhibits, I think we're entitled to
21 have --

22 MR. LEDBETTER: Okay.

23 MR. SCOTT: -- all of those,
24 whether he's identified them or not.

1 MR. LEDBETTER: I'll give them to
2 you.

3 MR. SCOTT: Thanks. I'll stop writing.

4 BY MR. LEDBETTER:

5 Q Is chlorinated naphthalenes and that family
6 related to PCBs in any way, or chlorinated
7 biphenyls?

8 A I would say they are related in a very broad way,
9 as being members of a large family of chemicals
10 referred to as chlorinated hydrocarbons.

11 Q And there's a number of chlorinated hydrocarbons,
12 are there?

13 A Hundreds.

14 Q All have the common characteristic that they are
15 chlorinated?

16 A They're chlorinated, and they have carbon and
17 hydrogen also.

18 Q Okay. And that particular portion makes up the
19 entire 100 percent of Aroclor, right; those
20 chlorinations and carbon and whatever the other
21 chemical was? Three chemicals in PCBs, right?

22 A That is correct.

23 Q So, some of the other chlorinated hydrocarbons
24 can have additional things besides what PCB has?

1 A Yes.

2 Q Do they all have at least what PCB has?

3 A In terms of the chemicals, but the configurations
4 are different.

5 Q Where chlorine, for example, is placed and where
6 hydrogen is placed and that sort of thing?

7 A And the carbons to each other and so on.

8 Q Okay. Back to terminology again. We often use
9 the term "Askarel" in referring to certain
10 polychlorinated biphenyl mixtures.

11 Are the two terms synonymous; PCBs
12 and Askarels?

13 A At what point in time?

14 Q Let's say after final production of an Aroclor,
15 say, Aroclor 1260.

16 When you have the final product
17 in a liquid form, that's called Aroclor 1260, would
18 you call that an Askarel also?

19 A At the time PCBs were used in electrical
20 equipment, the electrical industry referred to
21 those materials as Askarel.

22 Q Okay. So, if I could go a step further, you've
23 identified, for example, Aroclor is a product
24 that goes into the fluid that goes into

1 electrical equipment?

2 A Yes.

3 Q In some cases, it can be the entire fluid?

4 A Yes.

5 Q Pyranol is one of the trade names that contains

6 Aroclor products?

7 A Yes.

8 Q And then there are other trade names that also

9 contain Aroclor products?

10 A Yes.

11 Q All of those properly would be termed Askarels?

12 A I don't know what they'd be called today.

13 Q Okay. The last you knew, they could be properly

14 termed Askarel?

15 A Yes.

16 Q Okay. Is there any particular significance of

17 the 1200 series of Askarels, other than it's just

18 a PCB?

19 A I'm confused by the question.

20 Q Okay.

21 A Significance in what manner?

22 Q Why Aroclors have the -- or at least the PCB

23 Aroclors have a 1200 series designation.

24 Was there any particular meaning

1 or designation of 1200?

2 A Yes. The 1200 referred to the biphenyl.

3 Q So, any time we have the 1200 Aroclor, whatever
4 it is, it's always going to be a chlorinated
5 biphenyl?

6 A Correct; a mixture.

7 Q Now, there were other series of Aroclors, one of
8 which I think was 4400.

9 Were there others that you're
10 aware of; other series?

11 A There's a 5400 and a 60 series.

12 Q Anything significant about the 44, 54 or 60
13 series, similar to what the 1200 means for
14 chlorinated biphenyls?

15 A The 4400 meant that it was a blend of
16 polychlorinated biphenyls and polychlorinated
17 terphenyls.

18 The 5400 series referred to the
19 polychlorinated terphenyl mixtures.

20 The 60 series referred to a blend
21 of polychlorinated terphenyls and monochloro
22 biphenyl.

23 Q Any other series that you recall?

24 A There's a 2500 series.

1 Q What was the 2500 series?

2 A It was also a blend of polychlorinated biphenyls
3 and polychlorinated terphenyls.

4 Q Any particular difference between those two?

5 A The difference between the 2500 series and the
6 4400 series is the ratio of the biphenyls to the
7 terphenyls.

8 In the 2500 series, it was
9 predominantly biphenyl.

10 In the 4400 series, it was
11 predominantly terphenyl.

12 Q Is the -- I would assume then from that, that the
13 2500 series is more akin to the 1200 series than
14 would be the 4400 series to the 1200 series?

15 A That's because it has more of the biphenyl, yes.

16 Q Do you recall roughly what portion it was of those
17 two, 25 and 44?

18 A No, I don't.

19 Q But 44 had less terphenyls -- I'm sorry, more
20 terphenyls than it had biphenyls?

21 A Correct.

22 Q Were there PCBs used in the chlorinated pesticides?

23 A Not that I know of.

24 Q As plant manager in Anniston from '65 to '70, did

1 you ever get involved in the promulgation of
2 rules and regulations at the plant as to handling
3 and safety of PCBs?

4 A The safety and handling of PCBs as it refers to
5 human health were developed by the medical
6 department.

7 I did not promulgate any of those
8 rules.

9 Q Okay. And in what form did those promulgations of
10 the medical department or Medical Director come
11 to you at the plant?

12 A Are you talking about the phraseology, or the piece of
13 paper that showed up?

14 Q Yes. Not -- I don't want a chemical description of
15 the paper, for example. Just what the paper is called
16 and what system was used to get that word to you.

17 A I can only speak for the period in which I was
18 familiar with. And that involved sending to the
19 medical department, that page out of the Standard
20 Manufacturing Process Manual or the Standard
21 Operating Instruction Booklet; sending those
22 pages that involved safety and handling to the
23 medical department, and asking them to update or
24 review and update because we were updating the

1 whole manual.

2 They would make the appropriate
3 changes and send it back to the plant. And the new
4 version, if revised, would be incorporated in the
5 revision that was issued.

6 Q Did they then take on a different directive kind
7 of number or some sort of thing, or did it just
8 become part of the operating instructions; the
9 Standard Operating Instructions?

10 A It became a part of the operating instructions.

11 Q Okay. So, any time that you needed to check on
12 the safety or handling, you could just go
13 directly to those documents which were retained
14 at the plant?

15 A Yes.

16 Q And somewhere along the way, you or perhaps
17 someone else, would make a decision that they may
18 need to be updated, and they would then be sent to
19 the Medical Director or the plant doctor?

20 A Yes.

21 Q Would they be routed to first the plant doctor
22 and then to the Medical Director?

23 A Generally, the plant doctor would review the
24 document. And then the author of the revision

1 would take the plant doctor's version and send it
2 on to the corporate medical department for their
3 review and comment.

4 Q When you say "the author," is that somebody other
5 than the plant doctor that would author the
6 change?

7 A I'm talking about the author of the manuals of
8 which this safety section is a page or three or
9 whatever is appropriate; or a section of it.

10 Q Okay. So, they're big, thick manuals and part of it
11 is safety and handling?

12 A That is correct.

13 Q Do you know who the author of those manuals were?

14 A It varied.

15 Q Any particular group or department that would
16 have the authorship responsibility?

17 A The process manual, the highly-technical book, was
18 assigned to the plant technical services
19 department, which was the engineering and
20 scientific group.

21 One of the engineers or chemists
22 would be the principal author, and he would get
23 assistance from anyone in the plant or in the
24 corporation, in review of the sections.

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1 Q Okay. How about the standard operating
2 practices?

3 A The standard operating practices were, wherever
4 possible, assigned to the department foreman.

5 Sometimes the foreman did not
6 have the skills to prepare written documents, and
7 it would then be assigned to the department
8 supervisor.

9 Q With these process manuals and also the
10 SOP manuals, final approval went through
11 corporate headquarters and the Medical Director?

12 A For that section that was relevant to the Medical
13 Director.

14 Q Okay. And the other things would go through
15 other technical people within their relevance?

16 A Correct.

17 Q Again, at headquarters, though, for final approval?

18 A Again, it depended on the sections.

19 Q Some could be done without corporate headquarter
20 approval?

21 A Yes.

22 Q Do you recall in this time period, ever having any
23 direct contact with corporate headquarters, the
24 Medical Director's library or some such thing,

1 that may have relevant articles or directives
2 regarding PCBs?

3 A I'm not aware of anybody at the plant consulting
4 those records.

5 Q Okay. So, generally, you would have your own.
6 And if there was a difference at headquarters,
7 you wouldn't use them; you weren't allowed to use
8 them. Is that the idea?

9 A Oh, no. I was allowed to use them, but I relied
10 totally on the professionals in St. Louis.

11 Q Okay. And that would be the Medical Director and
12 his staff that you've identified?

13 A Yes.

14 Q How many people were a part of the Medical
15 Director's staff, if you know, in the '65 to '70
16 period?

17 A Oh, the professionals included, in addition to
18 the Medical Director, three individuals.

19 Q And who were they?

20 A Melvin Wheeler, who was the manager of -- I have
21 forgotten his title, but he was the individual
22 primarily involved with toxicology; toxicology
23 testing. And he was assisted by Dr. William
24 Hunt, Doctor of Toxicology.

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1 There was a Mr. Jack Garrett, who
2 was involved with work-place exposure and
3 industrial hygiene matters.

4 In addition to those four
5 individuals, there were the typical secretaries.
6 And in addition to that, there were part-time
7 physicians and about four or so full-time nurses.

8 Q Do you recall a Dr. Johnson who worked with Dr.
9 Kelly?

10 A Dr. Johnson, as I recall, joined Monsanto after
11 1970, to the best of my knowledge.

12 Q Okay.

13 A He was assistant medical director.

14 Q At the time that Monsanto purchased the Swann
15 Chemical Company, which I believe was in 1935, do
16 you know if there came with that purchase, the
17 patents or other technical documents relating to
18 the manufacture of PCB?

19 A It's my understanding that yes, it did.

20 Q Did you ever review the patents or that sort of
21 technical literature at the time?

22 Let me ask that question a
23 little differently.

24 You didn't do it at the time of

1 the purchase, certainly, but at a later date, did
2 you review documents pertaining to the technical
3 literature that was available from Swann to
4 Monsanto in 1935?

5 A I reviewed documents of that vintage.

6 I don't recall whether I reviewed
7 them all or not.

8 Q Do you recall if the patent information indicated
9 any potential adverse effects of PCB?

10 A No.

11 Q Do you recall what the claims on the patent was,
12 or claims in the patents?

13 A As I recall, these patents had to do with
14 process; what techniques were used in the
15 process.

16 Q When Swann was purchased, do you know if the
17 management and employees that were there were
18 just taken over and became Monsanto employees at
19 the time?

20 A That is my understanding.

21 Q Do you know who the plant manager of Swann was at
22 the time?

23 A No.

24 Q Was Swann involved in the manufacture of other

1 things in 1935, or prior thereto; other things
2 than PCB?

3 A Yes.

4 Q Do you recall again what proportion of the other
5 things, compared to the manufacture of PCBs, Swann
6 was involved with pre-1935?

7 A PCBs were a small part of the overall. I do not
8 know the ratios.

9 Q Do you know if there had been any other American
10 manufacturers of PCBs?

11 A At what point in time?

12 Q Post '35.

13 A I had reports given to me that when Monsanto
14 withdrew from some of the PCB market, that
15 someone in the southwest was attempting to sell
16 PCBs.

17 Q Would that have been a patent infringement?

18 A No. The patents had run out long ago.

19 I reported this to the Federal
20 authorities. I never did get a report on whether
21 they succeeded in determining what the source of
22 PCBs were.

23 Q Is it your understanding, from '35 through the
24 last manufacture of PCBs by Monsanto, that they

1 were the predominant force in the market here in
2 the United States?

3 A Yes.

4 Q Do you have any knowledge as to how much of the
5 American market was supplied by Monsanto from '35
6 up through, say, 1977?

7 A Which market?

8 Q The American market within this country. How
9 much PCBs was supplied to the users within this
10 country?

11 A Of all PCBs?

12 Q Of all PCBs.

13 A All applications?

14 Q That's right.

15 A High 90 percent.

16 Q Okay. So, out there somewhere in the world,
17 people had come a little bit into the market and
18 produced some in this country, other than
19 Monsanto -- or not produced in this country, but
20 supplied it to the country?

21 A That's my understanding.

22 Q Do you recall some sort of episode bringing to
23 your attention potential environmental effects,
24 adverse effects of PCB, other than those that

1 you were already aware of through the operating
2 literature and the process manual that you had
3 described to us?

4 A Again, what year?

5 Q Sometime after the '65 to '70 period.

6 A Of course, after 1970, I was assigned full-time
7 to monitor such reports.

8 Q Okay. We haven't gotten into all that, but we
9 will in a moment.

10 A I was aware of reports alleging that PCBs were
11 involved or could have caused the migratory bird
12 problem in the Irish Sea, and the death of young
13 seals in the North Sea.

14 I was aware of reports referring
15 to the brown pelicans off the coast of California,
16 and their inability to reproduce.

17 This was in the early 1970 era.

18 Q Okay. And that's when you became in that
19 neighborhood, when you became manager of
20 environmental control?

21 A Yes.

22 Q You described a moment ago that your
23 responsibility was then more involved in
24 monitoring PCB activities, so to speak, in that

1 1970 to 1972 period.

2 Is that true?

3 A Yes.

4 Q Okay. What else was your job duties and
5 responsibilities, besides PCB, while you were
6 manager of environmental control?

7 A PCBs was the only issue I was assigned.

8 Q So, Monsanto created a special position just
9 purely for PCBs?

10 A Correct.

11 Q Was that the first time that position had been
12 filled?

13 A Yes.

14 Q And you remained in that position until 1972?

15 Well, let me re-ask that. You
16 remained manager of environmental control until
17 1972, but it sounds like you still remained in
18 St. Louis from '72 to '77?

19 A Yes.

20 Q Was that a similar sort of job; still involved
21 with PCBs?

22 A The responsibility for PCBs was given to me in
23 1970, and I retained it until February, 1976.

24 Starting in about 1973, I was

1 assigned other chemical products in addition to
2 PCBs.

3 Q So, you just broadened your responsibilities, but
4 you still had PCBs in -- post-'76?

5 A No.

6 Q Okay. I missed the date there, then.

7 A '76 was the last date in which I was involved
8 with PCBs.

9 Q Okay.

10 A But between the period of about '73 to '76, I not only
11 had PCBs, but had additional products assigned to me.

12 After 1976, I had a list of
13 chemicals for which I was responsible, excluding
14 PCBs.

15 Q Okay. When you were manager of environmental
16 control, you stayed in that position up through
17 '72?

18 A Correct.

19 Q Who replaced you in that position?

20 A No one.

21 Q The title changed?

22 A The title changed.

23 Q And you continued with PCB responsibility only
24 for a while, and then you took up other chemicals

1 in '73 or thereabouts?

2 A Correct.

3 Q Still retaining PCB responsibility?

4 A Correct.

5 Q Did you have a staff in that first 1970 to 1972

6 period?

7 A No.

8 Q Was there other lateral departments responsible

9 for another individual chemical other than PCB?

10 MR. SCOTT: Responsible in what

11 way?

12 MR. LEDBETTER: Responsible for

13 another chemical other than PCB; meaning somebody

14 lateral to him, in a similar department.

15 MR. SCOTT: Do you understand the

16 question?

17 THE WITNESS: I think I do.

18 MR. SCOTT: Okay. I don't.

19 THE WITNESS: My hesitation,

20 again, is what point in time?

21 BY MR. LEDBETTER:

22 Q We're talking 1970 to 1972.

23 A '72. There's always someone throughout the

24 corporation responsible for a product.

1 In every case but PCBs, they, in
2 essence, wore several hats. They might be a
3 research person that has this responsibility,
4 they might be a manufacturing manager,
5 or it might be a marketing manager, it
6 could be an engineering type; depending on the
7 product and the applications and many other
8 factors.

9 PCBs were different, in that I was
10 given the full-time assignment without having to
11 wear another hat for at least three years.

12 Q Okay. My question then is -- perhaps stated
13 negatively, we could get to it better.

14 In this period of 1970 to 1972, you
15 were responsible solely for PCBs. And there was
16 no one else that you're aware of, responsible for
17 any individual chemical only.

18 Is that true?

19 A Not that I know of. But that doesn't mean I know
20 it all.

21 Q Okay. All right. Do you have knowledge as to
22 why a separate department was created for only
23 PCBs in 1970?

24 A I have an understanding.

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1 Q What is that understanding?

2 A PCBs involved two distinct groups within
3 Monsanto; two separate entities in terms of
4 management and responsibilities.

5 One was the functional products
6 group and the other was the plasticizers group,
7 with a common product. And, in essence, in order
8 to let the left hand know what the right was
9 doing, I was appointed this assignment, to serve
10 as a communicating link between the left hand and
11 the right hand and the two research groups and
12 the two marketing groups and the two manufacturing
13 groups.

14 Q Were you involved in the -- during this time, '70 to
15 '72, were you involved in the promulgation of
16 directives or standard operating procedures
17 relating to the safe handling of PCBs?

18 A Yes.

19 Q In what way?

20 A I was one of several individuals who reviewed
21 proposed drafts.

22 Q Of those regulations? And you would give suggestions
23 or approvals on those proposed drafts?

24 A I would comment or make changes where I saw

1 appropriate.

2 Q Did you attempt to stay up on the literature and
3 research up to that date, '70 to '72?

4 A Yes.

5 Q And that would assist you in making your
6 recommendations in such things, to the operations
7 manual, to the process manual, if that came to you?

8 A Yes.

9 Q And other similar type regulations?

10 A Yes.

11 Q I understand that Monsanto uses a bulletin
12 system relating to its products.

13 Is that true?

14 A Yes, they do have bulletins.

15 Q Is bulletins separate from the process manual and
16 the standard operating instructions that we've
17 talked about?

18 A Yes.

19 Q What is the bulletin system, used at Monsanto?

20 What is that bulletin system,
21 if you could describe it in broad terms?

22 A I don't know that it's used throughout Monsanto.
23 I can only speak for some of the business groups
24 that I happened to be working closely with.

1 Q Certainly. With only your involvement; fair
2 enough.

3 A In this instance, they issued these bulletins
4 which were -- I would personally call them
5 advertising literature promoting the products.
6 Describing to a potential customer or a current
7 customer the properties of the products
8 available; the kinds of uses to which they were
9 put or could be used, sort of futuristic; the
10 types of packages that it was available in.

11 That's about all I can think of
12 at the moment.

13 Q Would bulletins contain instructions for use,
14 as opposed to you can use it as a plasticizer?
15 Do you tell them how to use it as a
16 plasticizer, for example?

17 A Some bulletins actually had typical recipes; so
18 many pounds of this ingredient and X pounds of
19 that and you heat it to a certain temperature
20 and you get this kind of a adhesive.

21 Some of those were available, yes.

22 Q Did bulletins also contain warnings to customers
23 as to potential adverse effects of the products?

24 A Some of them, yes.

1 Q Is there a single department or group that would
2 be responsible for developing product bulletins?

3 A Well, the initiative is taken by the marketing
4 department, but they can go anywhere in the
5 corporation for assistance, depending on the
6 topic they wish to discuss or to include in
7 their bulletin.

8 Q Is that still, to your knowledge - at least the
9 last you knew at Monsanto - a normal way of
10 providing product literature; through the
11 bulletins?

12 A That is -- I'm confused by the use of the word
13 "normal."

14 It is one of many ways to
15 communicate.

16 Q It was still being used, the last you knew?

17 A Yes.

18 Q Are bulletins retained at some specific level or
19 department at Monsanto?

20 A To the best of my knowledge, the corporate
21 library keeps copies.

22 Q Where is that located?

23 A At Monsanto's corporate headquarters in St. Louis.

24 Q And, to your knowledge, do they keep current and

1 outdated bulletins?

2 A That's my understanding.

3 Q Did you ever see bulletins relating to Aroclor
4 products?

5 A Yes.

6 Q Did you ever see bulletins relating specifically
7 to Aroclor 1260?

8 A I don't recall a bulletin specific to a
9 particular Aroclor.

10 Q Okay. Would bulletins discuss Aroclors in
11 general terms. Or would they, the ones that
12 you're familiar with, cover specific Aroclor
13 numbers?

14 A They would cover the entire Aroclor family,
15 including not only the PCBs, but the PCTs,
16 except for those bulletins that are specific to
17 dielectrics.

18 They would then include only the
19 PCBs plus the blended materials. And the
20 hydraulic fluids bulletins would include only the
21 PCBs.

22 The heat transfer bulletins, again,
23 would refer to the heat transfer fluids by their
24 trade names, which would limit them to PCBs.

1 Q Okay. When we're talking dielectrics, that
2 would cover those Aroclor compounds that you
3 talked about earlier that was used in a
4 dielectric application, correct?
5 A Yes.
6 Q So, it would talk about that group in general?
7 A Yes.
8 Q Would the bulletins talk about a specific trade
9 name, such as Pyranol?
10 A They would refer to the word Pyranol as well as
11 other brands or trademarks.
12 Q Okay. Somewhere in the history here, I got a little
13 bit confused.

14 After you were manager of
15 environmental control, which you indicated was
16 1970 through 1972, you then assumed manager of
17 product acceptability. But from '72 to '73
18 only, your exclusive responsibility was PCBs.

19 Is that a fair statement?

20 A That's a correct statement, yes.

21 Q Okay. Even though the -- so, it was just a name
22 change for that first year, '72 to '73?

23 A Roughly, yes.

24 Q And from '73 to '77, you had PCBs plus other

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1 chemicals under your jurisdiction?

2 A Yes.

3 Q You described earlier that sometime in the early
4 1970 period, I think, that you became aware of
5 studies involving claims that there was adverse
6 environmental impact of certain PCBs?

7 A Yes.

8 Q Do you recall which Aroclors those were
9 primarily that were presenting the problem?

10 A The articles referenced Aroclor 1254 and Aroclor
11 1260.

12 Q Was it -- did you come to understand why only
13 those particular items were -- or those particular
14 Aroclor products were mentioned?

15 A I have my understanding, yes.

16 Q What is that?

17 A The references to Aroclor 1254 and 1260 indicate
18 to me that the analyst is finding as he analyzes the
19 materials, PCBs that are generally associated
20 with those commercial trademarks, indicating
21 that the higher-chlorinated types of PCBs are
22 present in the environment, which further
23 indicates that they are survivors in the
24 environment, as distinguished from the lower-

1 chlorinated PCBs which, at that time, generally
2 were not found, but we knew that a considerable
3 amount had been distributed in commerce.

4 Q Referring to some of the Aroclors that you
5 mentioned earlier in the 1200 series, was there
6 any particular Aroclor group that was
7 manufactured in greater quantities than any
8 other, in the 1200 series alone?

9 A As I recall, Aroclor 1242 was produced in
10 greater quantities.

11 Q And was that true from beginning to end?

12 A No. I think it peaked in about the 1960s,
13 somewhere in there.

14 Q Backing up again, back to 1935, is that when Aroclor
15 started; in about '35? The Aroclor compounds?

16 A No. They were initially introduced in '29,
17 became commercial in 1930.

18 Q Okay. So, when Monsanto bought that group from
19 Swann, they also bought the name Aroclor?

20 A Yes.

21 Q Okay. And 1242, to your knowledge, was
22 manufactured even back in 1929 or '30?

23 A Yes.

24 Q How about 1254?

1 A Yes.

2 Q How about 1260?

3 A Yes.

4 Q And in the early years, was it your
5 understanding that 1242 was manufactured - at
6 least up through the mid-1960s or thereabouts -
7 in greater quantities than the others?

8 A No. The 1254 and 60 were the greater quantities
9 initially. 1242 evolved through the years to
10 become the leader in the late '60s.

11 Q Was there any particular advantage -- again,
12 we're talking in terms that these were generally
13 used for dielectrics, correct?

14 A Yes.

15 Q They had some other applications, too, but not
16 nearly to the degree of dielectrics; is that
17 fair?

18 A That's correct.

19 Q Is there any particular advantage that 1254 and
20 1260 have over 1242 in a dielectric use?

21 A It's my understanding that in transformers, the
22 1254 and 1260 were the preferred PCBs, when
23 blended with chloro benzenes.

24 It's also my understanding that

1 Aroclor 1242 was the preferred PCBs in
2 capacitors.

3 Q Okay. Now, let me just try to back up on the
4 first preference to the 1254 and 1260 being used
5 preferably in transformers, and you added the
6 caveat, if you will, "when blended with chloro
7 benzenes."

8 Why that caveat?

9 A They are too viscous when used alone. They must
10 be diluted with a thinner.

11 Whereas, some companies used 1242
12 without the thinner, which is the chloro
13 benzene, in transformers.

14 Q Okay. Is that GE that we referred to earlier;
15 the Pyranol?

16 A GE preferred 1254 and 1260.

17 Q Okay. And that would be, again, blended
18 generally with chloro benzenes?

19 A Correct.

20 Q Do you recall any specifics of the studies, where
21 it came to your attention that these higher
22 Aroclors, 1254 and 1260, were being found in
23 various places in the environment?

24 Do you recall any specific

1 studies? Authors, for example?

2 A This is in the early '70s, you say?

3 Q Yes. We're in the 1970 through '77 period, when you
4 were involved with PCBs.

5 A Oh. That full period?

6 Well, the initial work was done
7 prior to the '70s at the University of
8 Stockholm.

9 Shortly thereafter, there was
10 work done by a Professor Widmark and
11 Dr. Jensen, Soren, S-o-r-e-n, Jensen,
12 J-e-n-s-e-n; a Dr. Robert Risebrough at the
13 University of California, Berkley; a Dr. David
14 Peakall at the Cornell University; a Dr. Thomas
15 Duke at the Commercial Fisheries Laboratory at
16 Gulf Breeze, Florida.

17 I've forgotten the names of the
18 investigators, but the Warf Institute (phonetic)
19 in Madison, Wisconsin, was doing some work with the
20 fish in the Great Lakes and found the higher-
21 chlorinated PCBs; the laboratory at Duluth,
22 Minnesota; at that time, it was still part
23 of the Federal Water Pollution Control Agency --
24 Administration.

1 I can't recall any others at the
2 moment.

3 Q Did you or anyone at Monsanto, to your knowledge,
4 conduct any tests or have any conducted, to attempt
5 to verify those findings?

6 A We verified the methodology.

7 Q What do you mean by that?

8 A The analytical methods used to analyze these
9 samples.

10 They used at that point in time,
11 techniques that were highly sophisticated. So we
12 chose to verify the methodology. And once our
13 professionals in the analytical laboratory
14 determined that the methodology had the ability
15 to make these distinctions, there was no reason
16 to question the results of the findings.

17 Q Was that when the gas chromatograph was
18 used?

19 A Yes.

20 Q And that was the methodology that was verified
21 through Monsanto?

22 A It was gas chromatography verified with
23 mass spectrometry.

24 Q And that was able -- satisfied Monsanto that in

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1 fact you could determine that these higher
2 Aroclors were in the environment?

3 A Yes. It isn't only the instruments, but it's
4 the way the sample is prepared.

5 Q Did anyone in this period of time at Monsanto,
6 make a decision that it's probably not good that
7 this is being found in the environment, and to
8 the levels it was found?

9 A We were -- yes. We decided that this was a man-
10 made chemical that should not be out there.

11 Q Now, as I understand it, you had as part of your
12 duties and responsibilities at Monsanto in that
13 period, sole involvement with PCBs or
14 responsibility with PCBs. You interacted with
15 the world community, if you would; did you
16 not?

17 A Yes.

18 Q And that included Dr. Widmark and Dr. Jensen in
19 Sweden?

20 A I did not ever meet Dr. Jensen. I met Dr.
21 Widmark.

22 Q Okay. And you were actually in Sweden?

23 A Yes.

24 Q And you were in Europe?

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1 A Yes.

2 Q And you were in Japan?

3 A No.

4 Q You met with Dr. Risebrough, I think?

5 A Yes.

6 Q At the University of California, Berkley?

7 A Yes.

8 Q And did you play any part after those meetings

9 in recommending to Monsanto that some sort of

10 additional measures had to be taken regarding

11 these Aroclors?

12 A Yes.

13 Q And what did you do?

14 A Well, I supported the plan --or the proposal to

15 withdraw from those applications which led to

16 uncontrolled disposal into the environment or

17 exposure to the environment.

18 Q And --

19 MR. SCOTT: Wait a minute. Let's

20 see if the witness has finished his answer.

21 THE WITNESS: Yes.

22 MR. SCOTT: Okay.

23 BY MR. LEDBETTER:

24 Q What applications is that, that you supported to

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1 ban or discontinue? I didn't get your word
2 there.

3 A Discontinue the sales to those uses in which the
4 items that contained PCBs were discharged into
5 the environment rather rapidly and without control.

6 Q Okay. What do you mean -- first, what do you
7 mean by "rapidly;" discharging PCBs rapidly into
8 the environment?

9 A Well, an example I can give is the application
10 in which PCBs are used in the early versions of
11 carbonless copy paper, where the paper once used,
12 is pitched into the wastebasket.

13 Q Okay.

14 A It can find its way into the City dump or it
15 can find its way into a recycle plant where the
16 chemicals are stripped off the paper and flushed
17 into the local river.

18 Q Do you recall --

19 A That is a rapid introduction.

20 Q Okay. Referring specifically to carbonless copy
21 paper, do you recall when Aroclors were first
22 used by Monsanto in carbonless copy paper?

23 A Monsanto didn't use PCBs in carbonless copy
24 paper. They sold to others who incorporated it

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1 in their process.

2 Q Do you recall when Monsanto first did that?

3 A Early 1960s is as close as I can come.

4 Q And that was one of the applications that was
5 discontinued in about 1970?

6 A Discontinued by mid-1971 -- early 1971.

7 Q Of the PCBs manufactured, we've talked about the
8 dielectric use, which is the majority. How much in
9 percentage, would you say, of PCBs was
10 carbonless paper between early 1960s to mid-
11 1971?

12 A Oh, I don't remember. I'd have to see the
13 numbers again.

14 Q Would it be a fair guess that it might be under
15 one percent?

16 A If you take the full time span, yes.

17 Q What are some of the other applications that
18 were found to be one of those rapid
19 introductions of PCBs in the system?

20 MR. SCOTT: Rapid and
21 uncontrolled, I think was his testimony.

22 MR. LEDBETTER: Yes. We're
23 talking about both categories. The first one is
24 just rapid.

1 THE WITNESS: There was wide use
2 in special paints.

3 An example that comes to mind is
4 the traffic lane white and yellow paints supplied to
5 the environment. It erodes and eventually
6 ends up in the soil, or rain washoff or runoff.

7 BY MR. LEDBETTER:

8 Q Again, what time period was it that
9 Monsanto began providing PCBs for the
10 application to special paints?

11 A It would have to be after World War II. I'd say
12 the early '50s.

13 Q Okay. And then ending when?

14 A We ceased selling in 1970. August 30 was the
15 last date of sale.

16 Q In that period of time, early '50s through
17 August 30th of 1970, in talking yearly
18 production of PCBs, what portion of it was
19 related to specialty paints, would you suppose?

20 A I have no idea. Most of this was sold through
21 distributors. There is no record of that.

22 Q When we were talking that the majority of PCBs
23 manufactured would have been for dielectric use,
24 would it be fair to say that the specialty

1 paints would be something less than five percent
2 of total production?

3 A I have no idea.

4 Q Any other special applications that you recall
5 being terminated in about early 1970 or '71?

6 A Yes. The uses in industrial hydraulic fluids.

7 Q How long had those been in use at Monsanto; the
8 PCBs for that purpose?

9 A The PCB type hydraulic fluids, again, probably
10 in the '50s.

11 Q And terminated when?

12 A Started in 1970 and completed early '71.

13 Q Is it a fair statement that these applications
14 that you've indicated to me are, generally
15 speaking, applications that were developed later
16 in time, well after when Monsanto purchased
17 Swann?

18 A Yes.

19 Q The primary use back in that time period was
20 what for PCBs; '35 up through, say, the early
21 '50s?

22 A Dielectric.

23 Q And dielectric even after the '50s, although
24 there were additional applications, dielectric

1 still remained the majority of the
2 product?

3 A Yes.

4 Q Any other large applications of PCBs that were
5 stopped in this early 1970 period?

6 A There's the broad usage. I'm going to refer
7 to as plasticizer. These, as you recall,
8 are the materials that soften the plastics to make
9 them flexible. Many PCBs were used for that
10 purpose.

11 Q And that was stopped again in the early '70s?

12 A August, 1970, was the date of termination.

13 Q Any other application that might make up any
14 substantial PCB product that was stopped in the
15 early '70s?

16 A The only other use that I can
17 think of that I would consider substantial
18 was the beginning of termination of
19 sales to the heat transfer applications.

20 The beginning steps were
21 concentrating on those uses that were related to
22 food-related processus in animal feed, or anything
23 that could get into the food chain. And this
24 termination was extended later to all heat

1 transfer uses.

2 BY MR. LEDBETTER:

3 Q Okay. And these actions were taken in response
4 to the reports - and your verified reports -
5 that Aroclors were being found in the
6 environment?

7 A Yes.

8 Q Any other reasons other than that?

9 A The termination in food uses was the additional
10 findings in some of the human food chain.

11 Q What findings are you referring to in the human
12 food chain?

13 A There was an incident that occurred in Japan
14 where oil was being distilled using a heat
15 transfer system with PCBs in it. And the oil
16 was contaminated with PCBs. That's one
17 incident.

18 Another one occurred as a result
19 of a heat transfer system used to sterilize fish
20 meal. The system leaked PCBs into the fish
21 meal, the fish meal found its way into poultry
22 feed, and eventually ended up in chickens and
23 eggs.

24 Another incident occurred,

1 relating again to chickens, with the heat
2 transfer system associated with a bakery. And
3 then the dairy incidences with silo coatings, and
4 the presence of PCBs in milk.

5 Those incidences relating to the
6 human food chain led to our decision to get out
7 of the heat transfer applications associated with
8 food processing.

9 Q Okay. If we had production figures from the
10 1960s running into the 1970s, after these uses
11 that you've described for us have been
12 discontinued and we see a corresponding drop in
13 production of PCBs, would it be fair that the
14 remaining production was for dielectric use?

15 A Yes.

16 Q So that would give us an idea of how much total
17 production there was for dielectrics?

18 A Yes.

19 Q Was there -- I forgot to ask the other wing of
20 that problem that Mr. Scott mentioned a moment
21 ago.

22 First, those things went rapidly
23 into the environment, you've described.

24 Then you mentioned also those

1 that could not be controlled?

2 A Correct.

3 Q What do you mean by "cannot be controlled"?

4 A Well, in my mind, the hydraulic fluids is an
5 example of a use of PCBs in what could
6 idealistically be described as closed systems.

7 But in the real world, they are
8 extremely difficult to maintain because of the very
9 high pressures involved. And the pressures on
10 the user to keep his operations going encouraged
11 the leakage of PCBs into the plant sewers or
12 into the sewage system, out into the environment.

13 We felt that it was unrealistic
14 to expect that application to be controllable.

15 Q Okay. And when you're referring to hydraulic
16 fluids, they might very well be part of a
17 hydraulic system which could be enclosed, but
18 you're saying with the pressures, Monsanto
19 really can't control whether they're going to
20 get into the environment or not?

21 A Not only Monsanto, but our customers.

22 Q Okay. So, you also considered the ability of
23 your customers to do or not do what they should
24 do with your product?

1 A That was very key in our thinking, yes.

2 Q Did you provide warnings to your customers
3 regarding what they should or should not do with
4 their products in this time period?

5 A Yes.

6 MR. LEDBETTER: Mark these the
7 next numbers, whatever they are.

8 (Whereupon Deposition Exhibit
9 Numbers 6 & 7 marked by reporter)

10 BY MR. LEDBETTER:

11 Q I'll show you first what's been marked as
12 Deposition Exhibit Number 6, and I'll ask if you've
13 ever seen that document or facsimiles of it?

14 A I have seen copies of this before, yes.

15 Q Okay. Do you recall if that corresponds with
16 about the time period that Monsanto was becoming
17 aware of the environmental problems that were
18 reported by the various studies?

19 A Yes.

20 Q And subsequently thereto, Monsanto did in fact
21 with its customers, provide certain notifications
22 of termination of products and notified the
23 customers as to what they should do with the
24 products to ensure that they don't get in the

1 environment, correct?

2 A Well, we did not tell the customer how to

3 achieve it. We just cautioned them: Don't let

4 it escape to the environment.

5 Q Okay. I'll show you what has been marked as Number

6 7, and ask if you can identify that particular

7 document; if you've seen it or a facsimile

8 before?

9 A I have seen Exhibit Number 7 before, yes.

10 Q Okay. And what was the purpose of that

11 particular document, if you know?

12 A This was to inform the customers on record with

13 Monsanto of PCB products, the latest of

14 Monsanto's understanding of the PCB situation.

15 Q Okay. And that included the environmental

16 problems, right?

17 A Yes.

18 Q Were there any efforts to provide that sort of

19 mailing to customers, regarding Monsanto's

20 experience as to the safety and handling and

21 working around PCBs at the Monsanto

22 manufacturing plants prior to 1970?

23 A I'm not aware of any mailing of this type that

24 addresses that subject.

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1 Q Were there other methods that you were aware
2 of that Monsanto used to inform customers of the
3 safehandling practices that have been
4 identified in the process manuals and the
5 Standard Operating Instructions manuals?

6 A Yes.

7 Q How was that done?

8 A The primary method of communicating it was
9 person-to-person from Monsanto's sales
10 representatives and from the St. Louis marketing
11 staff.

12 Specific medical questions were
13 answered by the medical department
14 representatives.

15 Another method used was via
16 bulletins such as we described earlier, which
17 addressed sections in safehandling.

18 The primary method was the
19 person-to-person contact.

20 There were occasions when the
21 customers' representatives would visit a Monsanto
22 plant or home office in which these topics were
23 also discussed.

24 Q And would the information from the Standard

1 Operating Practices manual be provided to the
2 customer at that point in time?

3 A Yes.

4 Q Now, when we're talking customers, we're not
5 necessarily talking the end user of the product.
6 We're talking, for example, in dielectric terms,
7 the customer who's buying the fluid to be
8 enclosed in an electrical piece of equipment.

9 That's the customer, right?

10 A That is the normal customer, yes; Monsanto's
11 customer.

12 Q Okay. Would Monsanto attempt to pass on the
13 information to the -- the safehandling information
14 to the ultimate users of the product?

15 A No. Monsanto had no such list.

16 Q Are you aware if Monsanto ever suggested that a
17 warning campaign of sorts be applied by its
18 customers to the end customers or the end users?

19 A I'm aware of such a suggestion occurring after
20 1970. I do not know what happened prior to
21 that.

22 Q Okay. And in what form did that suggestion to
23 the customer take place after 1970?

24 A I know I personally have talked to

1 representatives of Monsanto's customers along
2 these lines. And it was finally documented in
3 at least two documents. One was the ANSI
4 guidelines on how to handle Askarels.

5 And prior to that was a Monsanto
6 booklet referring to the handling of Askarels.
7 This booklet was revised in the '70s, and I know
8 for sure personally we recommended they get
9 extra copies and share with their customers.

10 Q Do you recall the name or title of that booklet?

11 A This Monsanto booklet?

12 Q Yes.

13 A All I remember is the word Askarel in the title.

14 Q Okay. And you said it was amended or modified
15 in some way in 1970 or thereabouts?

16 A Yes.

17 Q Meaning it was in effect sometime before 1970?

18 A Yes.

19 Q Do you recall its original effective date?

20 A I have seen copies dated in the '60s. I do not
21 know when the very first version was published.

22 Q Did Monsanto provide warnings and warning labels
23 with its PCB products?

24 A Yes.

1 Q Do you know when that started?
2 A As best I can determine, it started with the
3 Swann Chemical Company.
4 Q Did you ever see any copies of warning labels
5 that Swann had used?
6 A Yes.
7 Q Do you recall what they said?
8 A Not the exact words, but it warned that it
9 contained chlorinated hydrocarbon; avoid
10 prolonged breathing and avoid skin contact;
11 words to that effect.
12 Q Now, you indicated that you were aware that ANSI
13 came out with a standard on the handling of
14 Askarels?
15 A Yes.
16 Q And you were on the committee, weren't you?
17 A Yes.
18 Q And, in fact, you were the chairman of the
19 committee, weren't you?
20 A Yes.
21 Q And you then took the committee comments and
22 developed, or at least in part, began developing
23 a standard for C-107?
24 A Well, I didn't make the C-107 comments. I'm

1 puzzled by your question.

2 Q Okay. You, as chairman of the committee,
3 gathered the comments and the input for the
4 eventual development of that standard, correct?

5 A Yes.

6 Q What role did you play as chairman?

7 A I called for the meetings. I appointed the two
8 subcommittees. I assigned them to their work
9 rooms, and then spent my time going between the
10 groups to see that they were working and we had
11 a target date to meet. I just kept pushing for
12 some results.

13 I didn't do any of the work.

14 Q Okay. So you guided them along the path, so to
15 speak?

16 A Yes.

17 Q Okay. Do you recall any labels being provided
18 by Monsanto indicating that PCBs were a
19 potential human carcinogen?

20 A No.

21 Q You never recall any labels of that sort?

22 A That's correct.

23 Q Do you recall any requirements that such a label
24 be used?

1 A No.

2 Q Were you a part of the NIOSH committee?

3 A I was part of a review committee for a NIOSH
4 Criteria Document.

5 Q And that's the document that -- if I can find
6 it, I'll show it to you.

7 MR. SCOTT: Let the record
8 reflect - although you may continue with your
9 questions - that I will object to any
10 questioning of this witness or any other witness
11 with respect to any alleged human carcinogenic
12 potential of PCBs, as being irrelevant to these
13 cases.

14 MR. LEDBETTER: Let's have this
15 one marked as Number 8, I guess.

16 (Whereupon Deposition Exhibit
17 Number 8 marked by reporter)

18 BY MR. LEDBETTER:

19 Q Sir, let me show you what's been marked as
20 Deposition Exhibit Number 8, and I would ask if
21 you can identify that particular document?

22 A This is a copy of a publication of the U.S.
23 Department of Health, Education and Welfare, put
24 out by the National Institute of Occupational

1 Safety and Health, and it's entitled Criteria for
2 Recommended Standard....Occupational Exposure to
3 Polychlorinated Biphenyls (PCBs).

4 Q Okay. And you were on the review committee for
5 that recommended criteria, were you not?

6 A I was.

7 Q Did you have input into what went in the
8 recommended criteria?

9 A My input amounted to a critique of a draft that
10 was given to the review panel.

11 Q And this is the final as opposed to the draft,
12 correct; the recommended criteria?

13 A It appears to me to be a final. I have not seen
14 a final form.

15 Q Okay. So, the only one that you saw was a draft
16 copy of what was to eventually be the
17 recommended criteria?

18 A Yes.

19 Q Do you recall any input into the suggested
20 labels to be used or distributed with PCBs?

21 A Not specifically, no.

22 Q Okay. Do you recall having any input into
23 developing a material safety data sheet
24 requirement to be used in conjunction with PCB

1 products?

2 A No.

3 Q Are you aware that there is a requirement or a
4 suggested criteria by the NIOSH document that
5 that material safety data sheet be used?

6 A I don't remember.

7 Q Do you know if such a material safety data sheet
8 is used or was used at Monsanto from 1977
9 forward?

10 A Yes.

11 Q Does that apply to -- let me drop back.

12 When did Monsanto cease their
13 production of PCBs?

14 A Production was ceased in July, 1977.

15 Q Okay. And as I recall, this NIOSH Criteria Document
16 was effective in September of '77?

17 A That's the date on the document.

18 Q Okay. Did Monsanto manufacture or supply PCB
19 products of any sort after July of 1977?

20 A They sold -- they terminated production in July
21 of 1977, and terminated sales in September of
22 '77.

23 Q So, as far as material safety data sheets go,
24 assuming there was such a requirement or

1 suggestion effective in September, 1977, it wouldn't
2 have affected Monsanto, because they had ceased
3 production, correct?

4 A Well, I suppose technically, yes, but we did
5 prepare one.

6 Q Okay. You prepared it for all Aroclors or
7 certain Aroclor products?

8 A It was for PCBs.

9 Q Okay. PCBs in general, as opposed to a specific
10 type of Aroclor or specific polychlorinated
11 product?

12 A Correct.

13 Q And do you recall in what form that material
14 safety data sheet took; whether it was a number
15 or assumed some other sort of title other than
16 Material Safety Data Sheet?

17 A No, just Material Safety Data Sheet; a multi-
18 page document.

19 Q What is contained on that document as it relates
20 to potential hazards, safety, handling, and that
21 sort of thing, if you're aware?

22 A I don't remember.

23 Q Do you recall in general terms what is contained
24 therein?

1 If you'd like to, you can
2 reference the manual. It does have a format; a
3 grouping.

4 A Well, I remember the format. I don't recall the
5 specific wording.

6 It again referred to prolonged
7 breathing and skin problems. I'd have to see a
8 current version to refresh my memory. I haven't
9 looked at one for years.

10 Q Okay. Is such a material safety data sheet
11 still maintained --

12 A Yes.

13 Q -- for PCB products?

14 A Yes.

15 Q Revised periodically?

16 A Yes.

17 Q We've talked about the inhalation potential
18 problem with PCBs. And you've indicated, I
19 believe, that the levels that were suggested by
20 the industrial hygienists - one a governmental
21 group, one a private group - were at certain
22 levels for 1242 and 1254.

23 Do you recall any specific length
24 of time that those levels were okay or any

1 quantitative values as to how long you could
2 breathe them at that level?

3 A It's the 8-hour time weighted average.

4 Q And that's over the course of a normal work day?

5 A Correct.

6 Q Do you ever recall reading anything about short-
7 term, but greater exposures, in inhalation; the
8 adverse effects of those?

9 A No.

10 Q How does the vapor of PCB get into the
11 atmosphere?

12 A In many ways. I can develop a scenario where
13 liquid PCBs are spilled on a hot surface,
14 generating a vapor.

15 That's what happened in a die
16 casting mill working foundry. The transfer
17 systems, if they should develop a leak, would
18 release a very high temperature material. And
19 upon release, vapors can be seen coming off that
20 spill.

21 In order to get obvious
22 vaporization, it requires high temperatures.

23 At room temperatures, the vapor
24 pressure of PCBs is so low that the limits

1 established by these industrial hygiene groups
2 would not be exceeded.

3 Q And you can say that, irregardless of the size of
4 the room or the ventilation of the room?

5 A The size of the room, the amount of PCBs present,
6 the temperatures in the room and the period.

7 If the room has been sealed for a long time, it
8 could get saturated with PCBs.

9 How long this could take would be
10 variable. But it's conceivable; possible.

11 Q Okay. And the higher the temperature of the PCBs,
12 obviously, the other parts of that formula can
13 go down?

14 The higher the temperature, the
15 more vapor that's going to be put in the room
16 generally, correct?

17 MR. DINON: I'm sorry. Did you
18 say in general?

19 MR. LEDBETTER: In general is
20 what I meant to say. I think I said
21 generally.

22 MR. DINON: Okay.

23 THE WITNESS: In general, the
24 answer is yes.

1 I would like to elaborate on that
2 answer.

3 BY MR. LEDBETTER:

4 Q Sure. Feel free to do so.

5 A The thought occurs to me that the amount under
6 the condition we described of a
7 closed room, the possibility of PCB vapors
8 accumulating to some detectible levels, that is
9 also influenced not by only the conditions we
10 described - size of room, amount of spill,
11 temperature, how tight it is, how long it's been
12 enclosed - but it's also affected by other
13 materials in the room.

14 Q Such as?

15 A Well, I'm thinking specifically of transformers
16 fluids that have chloro benzenes in them.

17 Chloro benzenes are much more
18 volatile. They would come off first. Their
19 presence in that air space would impact on the PCBs
20 that also would be present.

21 Q What impact would they have; the chloro
22 benzenes?

23 A They are present eventually in the proportions
24 of which they are present in the original liquid,

1 and in proportion to their volatility.

2 There's a mathematical relationship
3 that can be worked out as to how much chloro
4 benzenes will inhibit the vaporization of PCBs.

5 Q Let's say that it's roughly on a 63 to 37
6 percent relationship.

7 Are you aware of how you would
8 figure out how that much chloro benzene would
9 affect the vaporization of PCB?

10 MR. DINON: 63 to 37
11 percent of which?

12 MR. LEDBETTER: 63 PCB, 37
13 chloro benzene.

14 THE WITNESS: I'm aware of the
15 basic mathematics that would be involved. You'd
16 have to go to a physical chemist and his
17 computer for him to determine it for you.

18 But it's dependent on the
19 temperature, the total pressure in the room, the
20 amounts in the original liquid, and the vapor
21 pressures of each of those.

22 And there is an equation that can
23 be satisfied, and an answer come out telling you
24 how much benzene is in the air and how much PCBs

1 as related to the amounts in the liquid.

2 BY MR. LEDBETTER:

3 Q Does one have more odor than another?

4 A Oh, yes.

5 Q Which has more odor?

6 A The benzenes.

7 Q Of course, also in your formula, you'd have to

8 plug in the heat, the size of the room?

9 A Well, the vapor pressure is related to the

10 temperature.

11 Q Are you aware in this particular circumstance,

12 that what we're talking about is allegedly

13 liquid leaking from PCB transformers?

14 A Yes.

15 Q Have you any idea of the amount of liquid that

16 we're talking about over the period of time that

17 we're talking about?

18 A In a general way. It's small amounts. Visible,

19 but not pools of it.

20 Q If that amount does leak out of transformers, you

21 know, a leakage so that it is visible running

22 down the transformers and even puddling to

23 some degree on the floor, would that suggest

24 to you that that would increase the likelihood

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1 of PCBs or chloro benzenes being found in the air.

2 MR. DINON: Objection to form and
3 foundation.

4 It's vague. Running down, pools;
5 the quantity isn't identified. The other variables
6 that he was referring to haven't been enumerated
7 or identified, to allow him to answer relevantly.

8 MR. SCOTT: Can you answer the
9 question?

10 THE WITNESS: To a matter of
11 degree. Does it suggest they would be present?
12 It does suggest that.

13 The quantities, I would have no way
14 of knowing.

15 BY MR. LEDBETTER:

16 Q Are you aware of any air samples being taken in
17 and around these particular transformers?

18 A No.

19 Q Do you have some idea of the size of the room
20 that we're talking about?

21 A No.

22 Q Do you have an idea of the size of the
23 transformers involved?

24 A I think I do, yes.

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1 Q And what is your understanding of the
2 transformers?
3 A I understand that they are something like in the
4 neighborhood of 30 to 35-gallon units.
5 Q Okay. And when you're talking of 30 to 35-
6 gallon units, you're talking about the amount of
7 liquid in the transformers?
8 A That's my understanding.
9 Q Okay. Did you come to learn that a certain
10 amount of that had apparently leaked out of
11 those transformers in some period of time?
12 A Yes.
13 Q Okay. Do you have any idea as to how much total
14 volume had leaked out?
15 A No.
16 Q Do you know what size the room is?
17 A No.
18 Q Do you know what the operating temperature
19 extremes might be on these particular
20 transformers?
21 A No.
22 Q If liquid -- let me withdraw that and start
23 again.

24 Assuming that we have an Aroclor

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WATER_PCB-SD0000058917

1 1260, and assume further that it is chloro
2 benzene that it's mixed with, would the
3 viscosity be greater than that of water?

4 A Yes.

5 Q And any degree as to twice as thick or -- I don't
6 know how we might measure that. Any degree that
7 you can estimate?

8 A No. I couldn't guess to it, no.

9 Q It would depend also on how hot it is, wouldn't
10 it?

11 A Yes.

12 Q Assuming that that liquid does leak out of the
13 transformers at seals and/or gaskets of sorts - just
14 assume that to be true, that there is visual
15 evidence of liquid leaking from the
16 transformers --

17 MR. SCOTT: Liquid that contains
18 PCBs? Because there's no evidence that the
19 liquid that leaked from these transformers was
20 PCBs.

21 BY MR. LEDBETTER:

22 Q Just liquid leaking out of these transformers,
23 and that these transformers contained PCBs;
24 assume that to be true.

1 Would it be a fair statement that
2 if liquid at this viscosity could leak out of
3 the transformers through the seals, that that vapor
4 would also have a tendency to come out of the
5 same area?

6 MR. DINON: Objection;
7 foundation.

8 You've mentioned this viscosity. You
9 haven't identified the viscosity. You haven't
10 identified the temperature. You haven't
11 identified any of the factors that he indicated
12 were necessary to determine the existence or the
13 nature of the vapors.

14 MR. SCOTT: Can you answer the
15 question?

16 THE WITNESS: I can't.

17 MR. SCOTT: Fine.

18 BY MR. LEDBETTER:

19 Q Okay. Are you aware of transformers that
20 breathe? Have you ever heard that expression for
21 transformers?

22 A Yes.

23 Q Do you know what that means?

24 A I think I do, yes.

1 Q What does that mean to you?

2 A As temperature changes occur in a transformer,
3 the amount of void space will fluctuate as the
4 liquid expands and contracts.

5 In order for that space to be
6 filled without collapsing the unit or destroying
7 it, it has to breathe. It has to make up for
8 that shrinkage and loss of void space somehow.
9 So the units do breathe.

10 Q Does that suggest to you that somehow that vapor
11 comes from the transformers, or is exhausted
12 some way through the transformers?

13 A I don't know. There's a gas, but I don't know what
14 it is. Some units have nitrogen blankets; some have
15 air; some could be vapor. I don't know the system.

16 Q Are you aware of any studies involving samplings
17 of surfaces around where PCB products have
18 vaporized and settled?

19 A No.

20 Q You're aware of no wipe samples being conducted
21 or that sort of thing?

22 A I'm not aware of wipe samples relating to PCBs, no.

23 Q Well, what material are you aware of where wipe
24 samples have been taken of vapors settling?

1 A I'm aware of dioxins. That's the only wipe test
2 I'm aware of.

3 MR. LEDBETTER: Mark this as Number
4 9, I think.

5 (Whereupon Deposition Exhibit 9
6 marked by reporter)

7 BY MR. LEDBETTER:

8 Q I show you what's been marked as Deposition
9 Exhibit Number 9, and ask if you can identify
10 that four-page document?

11 A I recognize this four-page document.

12 Q Okay. And that was a document prepared by you,
13 was it not?

14 A Yes; prepared at my direction.

15 Q Okay. What was the purpose for that particular
16 document?

17 A The addressee, Mr. Albert, had submitted a list
18 of questions and asked for some answers to those
19 questions.

20 Q Okay. And you attempted to provide those answers to
21 his satisfaction in that particular
22 document?

23 A Yes.

24 Q Do you recall arriving at any conclusions about

1 the potential effects or hazards of PCB in the
2 letter that you passed on to Mr. Albert?

3 A I don't know that I arrived at any conclusions,
4 no.

5 Q Do you recall what you informed him of, as to
6 potential hazards?

7 MR. SCOTT: Well, the document is
8 the best evidence of its own content,
9 counsel.

10 MR. LEDBETTER: Well, I'm giving
11 him time to review the document to see if he can
12 refresh his recollection.

13 MR. SCOTT: That doesn't make it
14 proper for him to testify with respect to it.

15 Go ahead.

16 THE WITNESS: As I indicated
17 earlier, this was prepared at my direction. The
18 answers to the questions were prepared by Mr.
19 Wheeler in the medical department. I did not
20 want to play doctor or toxicologist.

21 I then prepared the cover
22 letter to forward it to Mr Albert.
23
24

1 BY MR. LEDBETTER:

2 Q Okay. So, you're saying the inquiry came to
3 you, but the specific answers that were provided
4 to the specific questions of Mr. Albert were
5 provided by Mr. Wheeler?

6 A Correct.

7 Q And that was all sent out as an entire document
8 under your signature?

9 A Yes.

10 Q Do you recall in this time period that you're
11 involved in PCBs from 1970 to 1976, if you
12 were aware of any other studies listing
13 potential adverse effects of PCB?

14 A Other studies other than what?

15 Q Of other potential adverse effects.

16 We've talked about some of the studies
17 already, pertaining to dermatitis and chloracne
18 problems, correct?

19 A Yes.

20 Q We've talked about some studies relating to
21 potential respiratory problems, correct?

22 A Yes.

23 Q You've mentioned in passing a few minutes ago, that
24 you were aware of the Yusho incident that

1 occurred in Japan in 1968?

2 A Yes.

3 Q Were you aware, from that study and others, of other
4 potential harmful effects to those people from PCB?

5 MR. SCOTT: At what period of
6 time?

7 MR. LEDBETTER: This is from the
8 time period that he was involved with PCBs from
9 1970 through 1976, if he learned of other
10 studies.

11 MR. SCOTT: All right.

12 THE WITNESS: Let me try to recall.

13 Monsanto had under way studies
14 with rats, dogs and chickens in 1970. And these
15 studies were finally completed in '71.

16 I was informed that from these
17 studies, the following effects were noted: That
18 the higher-chlorinated PCBs as represented by
19 Aroclor 1254 and 1260, at the higher dose levels,
20 which was represented by the 100 parts
21 per million in the daily diet of the test
22 animals, resulted in loss in weight
23 gain of the test animal, enlarged livers with
24 some cell deformations, and some effect on the

1 reproduction in terms of size of the litters in
2 the rat studies.

3 In the chicken studies, the
4 lower-chlorinated PCB as represented by Aroclor
5 1242, affected the ability of the chickens to
6 reproduce in terms of hatchability of the
7 eggs and the thinness of the eggshell.

8 And this occurred at the low levels,
9 of about -- it was below 10 parts per million.
10 I think a retest showed 3 or 4 parts per million
11 in the daily diet.

12 Those are the Monsanto studies.
13 There was later another study using rats,
14 conducted by Dr. Renata Kimbrough of the CDC, in
15 Atlanta, Georgia. And her pathologist described
16 the liver cell changes as pre-cancerous.

17 I'm aware of the dialogue that
18 took place between the pathologist. I'm aware
19 that they looked at slides from the animal tissues.

20 And in fact with the Monsanto
21 studies, we went back to the reserved frozen samples
22 and sliced some more sections to make sure that the
23 damaged part of the liver was missed.

24 To make a long story short, the

1 conclusion was amongst the pathologists, that
2 the effects seen in the animals tested by Dr.
3 Kimbrough did show a change in cells. That
4 change was not observed in the animals tested by
5 the Industrial Biotech laboratories for Monsanto.
6 And there was an agreement in that amongst the
7 pathologists. The interpretation of the damaged
8 cells in Dr. Kimbrough's samples remains in
9 dispute, as I understand, to this day.

10 I'm also aware of a later study
11 by the National Cancer Institute, using Aroclor
12 1254. This now gets into the '80s.

13 The institute reported the
14 findings on that study, which was also a
15 lifetime study, that Aroclor 1254 did not
16 show cancerous effects.

17 I'm aware of some tests done in
18 the Gulf of Mexico by the laboratory at Gulf
19 Breeze, Florida, which established that shrimp, the
20 juvenile, the young shrimp, are extremely
21 sensitive to PCBs. They do not survive,
22 whereas the adult shrimp seem to be able to
23 withstand a fairly high dose.

24 I'm also aware that the Pautaxant,

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1 Maryland laboratory - the wildlife laboratory -
2 established earlier reports by Dr. Risebrough on
3 the pelicans in California attributing harm by
4 PCBs were not supported by the data. In the opinion
5 of the Pautaxant, Maryland laboratory, the DDT was
6 the cause for that affect.

7 The reports that were earlier out of
8 the North Sea and the Irish Sea - those reports
9 were finally published in, I believe, '72 -
10 indicated that the seals, the young seals died
11 because of malnutrition, separation from their
12 mothers.

13 The birds, as I recall, were
14 attributed to some storms and they were blown
15 off their normal paths and had nutrition
16 problems as well.

17 So, the presence of PCBs in those
18 animals was very real, but the effects were
19 attributed to other factors other than PCBs.

20 On the dog studies - going back to
21 Monsanto studies - the dogs were reported at
22 high levels of the high-chlorinated PCBs, to
23 suffer ulcers and bleeding in their digestive system.

24 I can't recall the other studies.

1 BY MR. LEDBETTER:

2 Q Okay. Referring specifically to the potential
3 liver effects of polychlorinated biphenyls, your
4 studies agreed, did they not, that PCBs, at least
5 as tested, were responsible for liver lesions?

6 A I'm not familiar with the lesion terminology. I
7 do know that there was an effect on the liver.

8 Q You also mentioned that there was an enlargement
9 problem that could be encountered with exposure
10 to PCB, correct?

11 A Correct.

12 Q How about dysfunction of the liver in some
13 way?

14 Are you aware of any problems of
15 that sort?

16 A I was led to believe that at very high levels
17 again.

18 Dr. Kelly's the best one to answer
19 that one on the affects and how the liver produces
20 certain enzymes in order to cleanse the body of
21 these materials.

22 Q Have you read any studies regarding cirrhosis of
23 the liver caused by PCBs or suspected to be
24 caused by it?

1 A I have not seen any.

2 Q Have you read any studies on the effect of PCBs
3 being related to impotence in human beings?

4 A No.

5 Q Have you read any studies regarding PCBs and its
6 effect in elevated triglycerides?

7 A No.

8 Q Have you read any studies regarding the
9 suspected relationship between PCB and a phenomenon
10 referred to as peripheral neuropathies?

11 A No.

12 Q In this period of 1970, you indicated that
13 Monsanto performed some studies.

14 Why were those studies performed
15 in 1970, if you know?

16 A With the reports where we later confirmed that PCBs
17 were being found in the environment and the
18 early allegations that they could be harmful,
19 Monsanto wanted to establish could they or could
20 they not harm living creatures.

21 There was no protocol for testing
22 for that purpose for industrial chemicals. Our
23 medical department sought advice from regulatory
24 agencies, which was not forthcoming. So they

1 chose to test these industrial chemicals in much
2 the same way that food chemicals are tested; not
3 with the intent of registering them for food
4 use, but to use existing test protocols.

5 In addition to testing with
6 mammals and birds, the initial plan included
7 testing with fish to establish aquatic toxicity.

8 The whole purpose of that was to
9 determine once and for all: Do these man-made
10 chemicals create any problems with living
11 things?

12 Q Were the fish studies done at Monsanto's direction?

13 A They were attempted. They proved a failure. And
14 by the time we decided that, our laboratory just
15 couldn't do it properly for us, there were
16 enough government laboratories involved.

17 And we decided that their expertise was
18 adequate, and any findings coming
19 from those labs were good enough for Monsanto.

20 Q Okay. The laboratory that you're referring to
21 is IBT?

22 A Yes.

23 Q Do you recall who it was that commissioned IBT
24 to do those particular studies?

1 A It was Monsanto, through Mr. Wheeler.
2 Q And Mr. Wheeler was part of Mr. Kelly's
3 department, right?
4 A Correct.
5 Q And they were commissioned sometime in 1970, and
6 concluded in '71?
7 A No. The studies were started in '68. There's
8 always some preliminary work that must go on to
9 establish feeding levels and protocols. The
10 lifetime feeding studies were terminated in '71,
11 which meant that they were started in '69.
12 Q So, the time period in '68, as I recall, that
13 was before there was the generalized knowledge of
14 the problem in the environment; is that true?
15 A Well, the confirmation of it. It was an evolving
16 situation. Monsanto started these early studies
17 quickly.
18 Q Okay. And was that after the Swedish doctors
19 had done their work?
20 A Yes.
21 Q And that, you believe was about 1968, when it was
22 first reported?
23 A In '67. And in '67, we had also sent samples to Dr.
24 Risebrough. And in '68, he was working in his

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1 laboratory and Mr. Wheeler was working with some
2 preliminary animal studies.

3 Q Was the test given to IBT in any way related to
4 the results of the Risebrough studies?

5 A The connection between the pelican and its egg
6 problems was considered when the dialogue with
7 IBT initially took place.

8 The intent there was to get IBT
9 to do some studies with some wild bird species.
10 They were looking at the Mallard duck. But by
11 the time the protocols were established and the
12 agreements made, the opportunity for getting
13 wild duck eggs had passed. They're not like
14 domestic chickens that lay eggs any time man
15 wants them to.

16 So, really as a substitute, it
17 was decided to go with the standard chicken test
18 and forego the wild duck test.

19 So, in answer to your question:
20 Yes. The work of Risebrough did trigger this
21 line of thought.

22 Q Okay. And eventually IBT ended up using
23 chickens as opposed to wild game?

24 A Correct.

1 Q And IBT's test of fish, you've indicated also
2 ended up -- or potential test of fish, also ended
3 up as a failure?

4 A Correct.

5 Q So, no evaluation out of that?

6 A Correct.

7 Q Subsequently -- well, first, who
8 is it at IBT that was involved in these
9 particular studies?

10 Was it one particular area over
11 there that the studies were given to at IBT?

12 A I don't think I understand the question.

13 Q Okay. Under whose jurisdiction were these tests
14 done at IBT, if you know?

15 A I can give you my understanding.

16 Q Okay. Dr. Calandra, the head of the laboratory
17 was involved. His principal assistant, and I
18 think he's the assistant director of the lab,
19 Dr. Fancher, was very much involved. There was a
20 Dr. Keplinger, who assumed the lead role when
21 Dr. Fancher retired.

22 And there were many other
23 individuals throughout the organization that did
24 the on-hands work in the laboratory.

1 Q Do you recall a fellow by the name of James
2 Plank (phonetic)?
3 A No.
4 Q Do you recall a fellow by the name of Dennis
5 Lindbergh (phonetic)?
6 A No.
7 Q Other than its connection to IBT, has Monsanto
8 had any connection with Dr. Fancher or Dr.
9 Keplinger, other than at IBT; any employer or
10 employee relationship?
11 A The only contact that I know Monsanto had was as
12 IBT employees, as a retiree of IBT, Dr. Fancher.
13 Q Okay. And the same with Dr. Calandra? He's
14 never been employed by Monsanto?
15 A That's true.
16 Q And James Plank you don't know?
17 A I don't know.
18 Q And you don't know Dennis Lindbergh?
19 A No.
20 Q How about Paul Wright? Do you know Paul Wright?
21 A I know Paul.
22 Q Who is Paul Wright?
23 A Paul Wright is an ex-employee of Monsanto and an
24 ex-employee of IBT.

1 Q Do you recall when Mr. Wright began working at
2 Monsanto?
3 A No.
4 Q Do you recall when he began working at IBT?
5 A No.
6 Q Do you know what time period that he worked at
7 IBT?
8 A No.
9 Q Do you know if he worked for Monsanto before and
10 after he worked at IBT?
11 A Before and after, yes.
12 Q He did so?
13 A Yes. It's my understanding.
14 Q If I gave you the period of, say, 1971 to 1972,
15 that Mr. Wright was at IBT, would that seem
16 about right to you?
17 A That's about right, sure.
18 Q And after he finished at IBT, he returned to
19 Monsanto at a different post than he had
20 previously occupied?
21 A Yes.
22 Q Do you recall what kind of post that was; what
23 position?
24 A He came in as a toxicologist reporting to Mr.

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1 Wheeler in Monsanto's medical department.
2 Q Was that originally, or after his stint at IBT?
3 A After.
4 Q Okay. Do you recall that IBT has been
5 criticized somewhat for the laboratory results
6 and the processus followed?
7 MR. SCOTT: In what situation,
8 counsel?
9 MR. LEDBETTER: Any time.
10 MR. SCOTT: Any investigation of
11 any kind?
12 MR. LEDBETTER: Any
13 investigation.
14 MR. SCOTT: Not necessarily the
15 one that Monsanto commissioned?
16 MR. LEDBETTER: Not necessarily
17 this one. Any one.
18 MR. SCOTT: Okay.
19 If you know, you can answer.
20 THE WITNESS: I'm aware of that,
21 yes.
22 BY MR. LEDBETTER:
23 Q Okay. And you're aware that criminal
24 prosecutions came from those?

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1 A Yes.

2 Q And you're aware that there was a conviction?

3 A Yes.

4 MR. SCOTT: Objection. You know,
5 the whole line is irrelevant, because, A, it doesn't
6 relate to any studies that Monsanto did.

7 And, B, it's totally irrelevant.

8 MR. LEDBETTER: I certainly think it
9 applies to the credibility of the studies done by IBT,
10 which is why I'm asking the questions.

11 MR. SCOTT: Oh, horse manure.

12 BY MR. LEDBETTER:

13 Q Were you aware of the conditions that allegedly
14 led to the complaints against IBT?

15 A I did not personally see them, no.

16 Q Okay. You had some knowledge of what was
17 happening at IBT that caused the grand jury to
18 indict certain members of IBT?

19 A I've heard allegations.

20 Q Did that affect -- to your knowledge, were those
21 allegations relating to the test processus being
22 followed?

23 MR. SCOTT: Again, counsel, in
24 what respect?

1 I think the question is vague,
2 it's improper in form, and I object to it unless
3 you specifically relate it to any test or any test
4 procedure so that this witness knows what you're
5 talking about.

6 BY MR. LEDBETTER:

7 Q Were you aware of what specific tests were
8 involved in the indictment?

9 A No.

10 Q Are you aware of what products were involved in
11 the testing?

12 A Some of them.

13 Q Are you aware if it was animal testing that was
14 being done?

15 A Yes.

16 Q Was it involving mice, for example -- or rats, I mean?

17 A I understand rats were mentioned.

18 Q Did it ever involve chickens?

19 A I don't recall chickens.

20 Q Do you recall anything about dogs or other primates?

21 A No.

22 MR. SCOTT: Wait a minute. Did you
23 say --

24 MR. LEDBETTER: Dogs or primates.

1 MR. SCOTT: Okay. Not "other"
2 primates.

3 Go ahead.

4 BY MR. LEDBETTER:

5 Q Do you recall if any of the tests they were
6 criticized for involved dogs or primates?

7 A No, I do not.

8 Q Did you ever hear of something called the Swamp
9 over at IBT?

10 A Only what I read in the Wall Street Journal.

11 Q Do you know somebody by the name of E. V. John,
12 J-o-h-n?

13 A Yes.

14 Q Who's Mr. John?

15 A He was a Monsanto employee working in the public
16 relations department.

17 Q Do you know what he did in public relations?

18 A I can't speak for his entire Monsanto career.

19 Q Just what you're aware of.

20 A He was assigned to work on public relations
21 matters associated with the organic division of
22 Monsanto Company in the period 1970, 1971.

23 Q So that was in the period that you were in
24 charge of PCBs, right?

1 A When I was initially placed in charge, yes.
2 Q Was he one of the fellows who had been
3 involved in getting information out to the
4 public about PCBs?
5 A Yes.
6 Q Do you recall somebody by the name of Howard Minkler?
7 A Yes.
8 Q Who is Mr. Minkler?
9 A A former Monsanto employee who in 1970, was the
10 general manager of the organic division.
11 Q And, again, the organic division included the
12 manufacture of PCBs, correct?
13 A Correct.
14 Q And under that division, as I recollect, came the --
15 I think it was called special fluids?
16 A Functional products group.
17 Q I'm sorry. Was there also a title called
18 functional fluids, or is that synonymous with
19 the one that you just mentioned?
20 A They're synonymous. Functional fluids group was the
21 correct title, I believe.
22 Q Okay. And then would that fall directly under
23 the organic chemical division?
24 A Yes.

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WATER_PCB-SD0000058940

1 Q Anything in between the two, as far as a step
2 up or step down?
3 A There's a position in there, but not a group. There's
4 an Assistant General Manager between the two.
5 Q Okay. But he would be Assistant General Manager
6 of the organic chemicals division?
7 A Correct.
8 Q And then reporting to him, I assume, would be
9 like the head of the functional fluids group?
10 A Yes.
11 Q Okay. Do you recall some fellow by the name of
12 Springate, J.E. Springate?
13 A Yes.
14 Q Who is Mr. Springate?
15 A Mr. Springate was the Director of the
16 plasticizers group.
17 Q Do you know if he had any involvement, to your
18 knowledge, with PCBs?
19 A Yes.
20 Q To PCBs contained within plasticizers?
21 A Yes.
22 Q Did he play any role in the literature that was
23 released to the public regarding -- I'm sorry, to
24 the customers regarding the use or cautions of

1 PCBs in plasticizers?

2 A He saw versions of the drafts as the document was

3 being prepared. He was aware that his group, his

4 directors of marketing were involved, and

5 supported that action.

6 Q Are you familiar with Orville Hicks?

7 MR. SCOTT: Orville Hicks?

8 MR. LEDBETTER: Orville Hicks.

9 THE WITNESS: Yes, I'm familiar with

10 Mr. Hicks.

11 I've forgotten what his assignment

12 was.

13 BY MR. LEDBETTER:

14 Q Something in chemistry?

15 A I associated him with the research department.

16 Q A research chemist?

17 A Yes.

18 Q Did he do any work on PCBs that you're aware of?

19 A As best I recall, yes. But I don't recall

20 specifically what.

21 Q Would he have done some of the analyses that you

22 were talking about earlier to verify the content

23 or the purity of PCBs, for example?

24 A I do not associate Mr. Hicks with that type of

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1 work, no.

2 Q Do you recognize the name Cumming Paton?

3 A Yes.

4 Q What was Mr. Paton's job duty or job duties?

5 A Of course, it varied through the years.

6 He was a marketing representative
7 of the plasticizers group in 1970. He then
8 became the principal PCB type plasticizer spokesman.

9 Q Okay. Were you aware if he had authored or at
10 least sent through his office various
11 communications to customers regarding PCBs or
12 their safe use of PCBs?

13 A Yes.

14 Q And your understanding is that was primarily in
15 plasticizers?

16 A Yes.

17 Q How about a fellow by the name of Pickard? Do
18 you know a Mr. Pickard?

19 A Pickard?

20 Q That you may have gone to Washington with at some
21 time?

22 A I'm sorry. I was thinking of the wrong spelling.
23 Sam Pickard.

24 Q And what did Mr. Pickard do?

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WATER_PCB-SD0000058943

1 A Mr. Pickard is the Vice-President in charge of
2 the Monsanto Washington, D.C. office.

3 Q What kind of office does Monsanto have in
4 Washington, D.C.?

5 MR. SCOTT: What do you mean, what
6 kind?

7 BY MR. LEDBETTER:

8 Q What do they do there?

9 A Some of it is lobbying efforts. And a good bit of
10 it addresses the registration of agricultural
11 chemicals, pesticides, weed killers and the like.

12 And also they handle the
13 registration of approved chemicals with the Food
14 and Drug Administration.

15 Q And did --

16 A And also contacts with the Environmental Protection
17 Agency, and other tax departments, and commerce and
18 so on.

19 Q Did you have the occasion with Mr. Pickard to
20 accompany him or he accompany you to meet with certain
21 legislators regarding the question of PCBs?

22 A No. Mr. Pickard and I did not go together.

23 Q Do you recall accompanying him elsewhere, other
24 than to Washington, for a subject concerning PCBs?

1 A I do recall I accompanied Mr. Pickard on other
2 matters. I don't recall any with PCBs.
3 Q Do you know a Paul G. Benignus?
4 A Do I know him? Yes.
5 Q What does Mr. Benignus do or what did he do?
6 A Mr. Benignus was a former Monsanto employee who
7 was the marketing manager for dielectric fluids.
8 Q Do you remember what time period that was?
9 A Gosh, almost his entire career.
10 Up until he retired in 1972.
11 Q What about Cole Webber? Who is Mr. Webber?
12 A Mr. Webber was in 1970, a manager of product
13 acceptability, who assumed PCB responsibilities
14 in 1976 when I was re-assigned.
15 Q Did he work with you prior to 1976?
16 A On PCBs?
17 Q Yes.
18 A For about three months we worked together, yes.
19 Q And that was just prior to your leaving that position
20 in 1976?
21 A Correct.
22 Q And he succeeded you then in the position with
23 responsibility of various chemicals, including
24 PCBs, in that time period, 1976?

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1 A That is correct.

2 Q Do you know how long he remained in that position?

3 A No, I do not.

4 Q Do you recall any information that you provided
5 to Mr. Webber regarding PCBs, or access to
6 information?

7 A I gave him my whole set of files; everything.

8 Q You had separate individual files at that time,
9 in 1976?

10 MR. SCOTT: That isn't what he said.

11 THE WITNESS: I had PCB files that
12 I turned over to Mr. Webber.

13 BY MR. LEDBETTER:

14 Q Okay. Can you describe those files to me; what
15 files you had that you turned over?

16 A As I recall, they were about four four-drawer types,
17 about 16 drawers full of paper, containing all of
18 the documents that I had accumulated since 1970.

19 Q Did this include studies, for example?

20 A Copies of them. The originals were over in the
21 medical department.

22 I had summaries and abstracts and
23 shortened versions; not the full documents.

24 Q Did you have the studies, for example, as far back as

1 the 1930s at that time period?

2 A There were some copies in there, yes.

3 Q And did you have copies of certain bulletins
4 regarding PCB products?

5 A Yes.

6 Q Did you have copies of certain Standard Operating
7 Practices Manuals, or portions of them?

8 A No.

9 Q How about process manuals or portions of those?

10 A No.

11 Q Any other category of information that you would have
12 retained in your 16 drawers of filing at that time,
13 from '70 through '76?

14 We've talked about studies, we've
15 talked about bulletins.

16 A Letters to customers, letters from customers,
17 responses, correspondence with government
18 agencies, government documents, copies of PCB
19 scientific literature, research reports that
20 related to PCBs, labels, copies of revised labels,
21 or correspondence relating to proposed changes in
22 labels, copies of presentations before Monsanto
23 committees or regulatory agencies, or even a
24 congressional committee.

1 Q Did you appear before any congressional
2 committees regarding the subject of PCBs?

3 A Yes.

4 Q Which ones?

5 A I don't recall its exact name. It was a
6 subcommittee that I recall was associated somehow
7 with Merchant Marine chaired by Congressman Stubbs
8 of Massachusetts.

9 Q Any others that you recall?

10 A Not congressional.

11 Q On your C.V., we had only gotten up to '72
12 through '76.

13 You remained in the same position,
14 although without responsibility with PCB, as I
15 recall, up through '77?

16 A That is correct.

17 Q And then in '77, you took a similar titled job, but at
18 a different location?

19 A No. What happened is that Monsanto reorganized
20 and formed a new unit.

21 Q And that's where the Intermediates comes in?

22 A The Chemical Intermediates Company.

23 Q And you did not have involvement with PCBs in
24 that time period?

1 A That is correct.

2 Q Were you periodically called upon to provide
3 service to Mr. Webber or advice to Mr. Webber or
4 others at Monsanto regarding PCBs?

5 A Not often, but I was available, yes.

6 Q Did you have occasion to involve yourself in
7 litigation through either providing support
8 services or being deposed or trial testimony?

9 A Yes.

10 Q Do you recall approximately how many times you
11 appeared at trial?

12 A In all or just '77?

13 Q From forward; 1977 forward.

14 A '77 forward --

15 Q Relating to PCBs; not anything else you may have been.

16 MR. SCOTT: To today?

17 MR. LEDBETTER: To today.

18 MR. SCOTT: This is one.

19 MR. LEDBETTER: Well, this isn't
20 a trial yet, but this is one.

21 MR. SCOTT: That's right.

22 THE WITNESS: I'm going to guess
23 somewhat here. Since '77, I've been involved
24 with litigation matters about eight times.

1 BY MR. LEDBETTER:

2 Q Okay. Now when we're talking eight times, is
3 this at trial actually, or is it depositions, or
4 just any litigation matter?

5 A One trial and seven depositions. This is an
6 estimate. I don't have a score sheet.

7 Q Okay. The trial that you appeared at, do you
8 recall where that was?

9 A It was up in Michigan.

10 Q And that's Bad Axe, Michigan?

11 A Yes.

12 Q And that was regarding silos or something, wasn't
13 it; the plasticizers on the inside of the silos?

14 A Yes.

15 Q The Aroclor involved there was a different
16 Aroclor than 1260 and the 1200 series, correct?

17 A It was a 1200.

18 Q Was it a 1200 series?

19 A Yes.

20 Q Okay. What were the seven depositions, if you
21 recall those; what they were involved with,
22 roughly?

23 A I'll try to remember them.

24 Depositions involved with the Michigan

1 case we just talked about.

2 I'm having trouble with my dates.

3 A deposition in South Carolina
4 involving contaminated waterways from a capacitor
5 plant.

6 A deposition relating to the
7 Bloomington, Indiana landfill and sewage
8 treatment being contaminated with PCBs from a
9 capacitor plant.

10 A deposition regarding the
11 replacement of transformers in an office
12 building in which the owner was seeking financial
13 relief.

14 There was a deposition regarding
15 some transformer manufacturer in Texas, who had
16 dumped PCBs in his property. A State agency was
17 seeking remedial cleanup action and he was
18 seeking financial reimbursement.

19 Last week I had a deposition in a
20 case in Texas, in which many individuals are claiming
21 health effects due to the exposure to PCBs at the
22 work place.

23 I don't recall any more, but --

24 Q Do you recall another one in Michigan, regarding

1 PCBs in some sort of dental compound?

2 A Yes. But that was before 1977.

3 Q Okay. You think that that position occurred sometime

4 before '77?

5 A I think it did.

6 Q Okay.

7 A But like I said, I'm having trouble with my dates.

8 Q Do you recall any involvement with any case in

9 California regarding PCBs?

10 A No, I've never been involved.

11 There was a case in Colorado.

12 Q Do you recall what that one was about?

13 A I take that back.

14 I was prepared to appear and I was

15 told to go home. So, I never did participate in

16 that one. I don't know what happened.

17 Q Okay. Do you recall the type of product involved

18 there?

19 A It was PCBs. I don't recall the source. But it

20 seems to me that PCBs somehow were found on a farm,

21 and the owner of the farm claimed some harm

22 to his property.

23 I don't know if it was heat

24 transferred PCBs or dielectric. Scrap PCBs

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1 is all I remember.

2 Q And then prior to 1977, other than the dental
3 case, do you recall any other PCB cases that you
4 were involved in with either a trial or a
5 deposition?

6 A Yes.

7 Q What were those?

8 A Well, there were two that went to trial.

9 One I will refer to as the mink
10 case in New Hampshire.

11 Q That was the claim about the mink somehow losing its
12 mink or something of that sort? The poor things
13 dying or some such thing?

14 A The allegation was that as a result of ingesting
15 PCBs in their diet, the mink had two problems.
16 One, they could not reproduce normally; they
17 failed to reproduce.

18 Secondly, their pelts were
19 inferior.

20 Q The second trial?

21 A The second trial was a trial brought in the State
22 of Tennessee, involving transformer liquid of a
23 new transformer on route from a GE plant to a
24 customer. It developed a leak. The driver of

1 the transfer vehicle was pulled aside by the
2 Highway Patrol. They talked to the dispatcher
3 and the dispatcher advised them to just drain the
4 whole unit. And he did, on the shoulder of the road
5 onto the farm land. And as a result, we had a
6 farmer claiming health effects for his family and
7 damage to his farm value, contaminating a cattle
8 pond and contaminating his herd.

9 In addition to that, there was an
10 individual who was on the same roadway heading in
11 the opposite direction, claiming that with his
12 window down, he was splashed with the material and
13 suffered harmful health effects.

14 Q Do you recall what time period that that first
15 alleged exposure occurred?

16 A I believe it was in '74, and the trial was '75;
17 something like that.

18 Q Did you give a deposition also in that case?

19 A It was a very short deposition and then we went
20 to the courtroom that same day.

21 Q All right. Any other depositions in that pre-
22 1977 period? We talked about the two trials.

23 A Yes. Both trials had depositions involved in
24 them.

1 Then there was a North Carolina
2 chicken case; Holly Farms Chickens. This had to do
3 with breeding hens not laying eggs, and the eggs that
4 were laid wouldn't hatch - it effected the whole
5 operation - as a result of contaminated poultry
6 feed.

7 There was a similar situation that
8 occurred in New York State in the Catskill area;
9 PCBs in poultry feed affecting flocks.

10 In Indiana, there was another
11 dairy case with silo coatings affecting cattle
12 herds.

13 In Montana, there was a case of a
14 leaking transformer in a packing house, - beef and
15 pork packing - where the transformer oil got
16 into the sewage system from which waste from
17 the packing operation are collected and salvaged and
18 placed in animal feed; you know, the fat and the
19 blood, along with PCBs.

20 That was in Montana.

21 Did I mention a case in -- yes, I did.

22 Q The Catskills?

23 A South Carolina.

24 Q South Carolina? I have North Carolina, Holly

1 Farms.

2 A Yes. Oh, yeah. The Waukegan Harbor-Outboard Marine.

3 This was the use of PCB hydraulic
4 fluids at Outboard Marine.

5 MR. SCOTT: Hole on one moment.

6 (Off the record)

7 BY MR. LEDBETTER:

8 Q We were talking a moment ago, Mr. Papageorge,
9 about the number of depositions you had given
10 pre-1977, and the last one you've discussed with us
11 was the Waukegan.

12 Is there another one after that, or
13 another one in that time period that you can recall?

14 A I can recall one more, but I don't recall which
15 time period it falls into.

16 This relates to PCBs in New
17 Bedford, Massachusetts Harbor.

18 Q That was reported, as I recall, in some of the
19 popular literature?

20 A I believe I saw some press clippings, yes.

21 This related to PCBs in capacitors.

22 Q Was there a manufacturing facility of capacitors
23 near there?

24 A Two of them.

1 Q Do you recall who those were?
2 A The Aerovox Company and Cornell-Dubilier.
3 Q Okay. Any other depositions or trial testimonies that
4 you can recall; other than those you've mentioned to
5 us?
6 A I think I've covered them all. I don't recall any
7 other ones.
8 Q We've talked about a number of records and
9 documents relating to PCBs.
10 Was there a record-retention
11 policy, or is there now, if you know, for
12 documents at Monsanto?
13 A Yes.
14 Q Is it a formalized document?
15 A Yes.
16 Q Do you recall what the retention policy is as to
17 those items relating to PCBs?
18 A Well, PCBs are not mentioned in the formal
19 program. My policy was to keep everything forever.
20 Q Do you know if that's been followed since you've
21 left your office there in 1977?
22 A I do not know.
23 Q Have you been involved with PCBs, other than this
24 trial testimony and depositions, since 1977,

1 in your job assignments at Monsanto?

2 A Yes.

3 Q Okay. Maybe we could get back to that real

4 quick.

5 A I'm sorry. I misled you. Not in my job assignment.

6 Q Your involvement has been something collateral to

7 your jobs?

8 A Correct.

9 Q Can you describe what that involvement has been,

10 other than depositions and trials?

11 A That's it.

12 Q Okay. In conjunction with this case, did you

13 review any documents relating to this case prior

14 to appearing here today?

15 A I was shown a group of documents that I was

16 informed had been involved in this case in some

17 manner.

18 Q Do you know what kind of documents those were or

19 what they concerned?

20 A I just can't distinguish one set of documents

21 from another anymore.

22 I really don't remember.

23 Q Do you recall if it was some compilation of

24 facts, for example, or if it was a deposition or

1 that sort of thing?

2 A No. It was, as I recall, a sample of a letter sent
3 to customers.

4 Q Do you recall the time period, the vintage of that
5 letter, sent to customers?

6 A It was in February, 1970; very much like the one
7 that was submitted as an exhibit here earlier.

8 Q This one here is dated February 18th, 1970. But
9 there's a similar letter that went out as
10 a mass mailing on February 7th, 1970?

11 MR. SCOTT: No, no, no, no.

12 THE WITNESS: February, 1970.

13 BY MR. LEDBETTER:

14 Q Okay. I'm sorry. In February, 1970, that kind of
15 letter went out to customers?

16 A Yes.

17 MR. SCOTT: That's the letter I
18 think he's talking about.

19 THE WITNESS: Yes.

20 BY MR. LEDBETTER:

21 Q Do you recall a specific letter to a specific
22 customer that you reviewed in this case?

23 A No.

24 Q Anything else in documents that you reviewed, that

1 you recall?

2 A I just can't recall.

3 Q How about photographs? Have you ever seen any
4 photographs relating to this case?

5 A No.

6 Q Never saw any photos of the transformers, for
7 example?

8 A No.

9 Q Or the transformer room?

10 A No.

11 Q Did you ever read any depositions or summaries of
12 depositions?

13 A No.

14 Q Or excerpts of depositions?

15 A No.

16 Q Did you read any analyses, lab analyses of
17 transformer fluid and/or tissue samples, blood
18 samples, or anything like that?

19 A No.

20 Q Did you discuss those with anyone?

21 A Mr. Caretti indicated that -- let me think a bit now.
22 I'm getting my cases confused again.

23 I remember a reference to some
24 analyses that questioned as to whether the right

1 chemical was analyzed.

2 Q Do you remember anything more about whether the
3 right chemical was analyzed?

4 A As it was described to me, the chemical was
5 listed as PBB. And through the B, the center B, a
6 C was written over it, creating some question as
7 to which chemical was analyzed.

8 That's all I recall of that discussion.

9 Q All right. Were you sent any documents relating
10 to this case?

11 A No.

12 Q None actually delivered to your hands or your
13 possession?

14 A That is correct.

15 Q Those documents you have reviewed has only been
16 while they're in the possession of someone else?

17 A Yes.

18 Q Has that someone else primarily been Monsanto's
19 attorneys?

20 A Yes.

21 Q Did you review documents in the hands of anyone
22 at Monsanto relating to this case?

23 A Not a Monsanto employee.

24 Q Okay. In 1977, you became environmental

1 operations Director, correct?

2 A Right.

3 Q What was your job duties as environmental
4 director?

5 A I had reporting to me a staff of professionals, and
6 we covered the three general areas.

7 One was involved with plant
8 emissions, solid waste, and the water affluent
9 issues.

10 The other was the work place
11 health issue; industrial hygiene kinds of
12 matters.

13 And the third was the product
14 acceptability function. This had to do with
15 Monsanto products after they left the plant gate
16 on route and in use.

17 Q Making sure they get there safe, so to speak?

18 A Get there safe. And we communicated to the
19 customers all we knew about it, and how they should
20 be handled and so on.

21 Q Was that involvement with PCB in that time
22 period?

23 A No.

24 Q Somebody else had responsibilities relating to

1 PCBs from '77, forward besides Mr. Webber?
2 A Oh, yes. It changed several times.
3 Q Do you recall who it was that succeeded Mr.
4 Webber?
5 A I believe the activities were assumed by Mr.
6 David Wood. But I was not close to that, so that's
7 an impression I have.
8 Q Do you recall when Mr. Wood may have assumed
9 those responsibilities?
10 A When?
11 Q From Mr. Webber.
12 A No, I don't.
13 Q Any others succeeding Mr. Wood that you recall?
14 A Mr. Wood was followed by Dr. John Craddock,
15 C-r-a-d-d-o-c-k, who currently has that
16 assignment.
17 Q And his assignment, much like your former
18 assignment, was in charge of PCBs and other
19 chemicals, too?
20 A No. As I understand it, it's exclusively PCBs.
21 Q Did the records retention policy that we
22 talked about, have a specific document name? That
23 multi-page document that pertains to records
24 retention, does it have a name or a number?

1 A It had a title. It was a booklet issued to all
2 employees, with the policy described and
3 guidelines, what documents should be addressed
4 and the retention time appropriate for each, under
5 many sub sections.

6 I don't recall the title of the
7 document, but I know it had the words record
8 retention program in it, and I know it had the
9 word Monsanto in it.

10 Now, the best I can recall at the
11 moment is Monsanto's Record Retention Program.

12 Q Do you recall if at any time while you were at
13 Monsanto, the PCB literature may have been
14 incorporated in a computer?

15 MR. SCOTT: Objection, counsel.

16 I think you're getting into a
17 privileged area and I'm going to instruct the
18 witness not to answer the question.

19 MR. LEDBETTER: You think my
20 question relating to whether it's put on a
21 computer is privileged?

22 MR. SCOTT: Yes. The attorney-
23 client privilege may be within that, and I'm
24 going to instruct the witness not to answer.

1 You can ask me the question if
2 you want, and if it isn't a matter of privilege,
3 then I'll give you an answer. And I'll make it
4 sworn.

5 MR. LEDBETTER: Can I do special
6 interrogatories to you in that regard?

7 MR. SCOTT: If you want to, yes.
8 But I'm going to instruct this witness not to
9 answer until I've had an opportunity to discuss
10 it with our client.

11 MR. LEDBETTER: Okay. And if it
12 is something that's discoverable, then maybe
13 special interrogatories can satisfy my inquiry.

14 MR. SCOTT: Fine.

15 Can you give me the question back,
16 Lori, or not?

17 THE REPORTER: Yes.

18 (Whereupon previous question read back)

19 MR. SCOTT: We can debate it later
20 and I'll make an inquiry for you and determine
21 whether or not I'm going to assert a privilege.

22 MR. LEDBETTER: Okay. And then,
23 of course, I'm going to --

24 MR. SCOTT: If I assert a

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1 privilege, I'll give you enough information to
2 argue your question.

3 MR. LEDBETTER: Fair enough. And
4 we can do some sort of an evidentiary hearing if
5 we need to.

6 MR. SCOTT: You can give me a
7 special interrogatory if you want.

8 MR. LEDBETTER: Okay.

9 BY MR. LEDBETTER:

10 Q At the time that you were aware of all the
11 literature in your office, was there an indexing
12 system that you kept?

13 A Do you mean was there a log kept on a separate
14 piece of paper somewhere?

15 Q Something that would lead you to whichever file drawer
16 it was in. You've got 16 file drawers. What's in
17 which.

18 A Very crude. Just open the file drawer and look
19 at the files and see what might fit.

20 Q And I remember I put it back here in 1972 or
21 something?

22 A And actually it evolved, and I became very dependent on
23 the secretaries to retrieve documents for me.

24 Q Do you have any knowledge if at a later time, an

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1 indexing system was ever established for the PCB
2 documents in your office?

3 A I do not have any knowledge.

4 Q How about at the Monsanto Medical Director's office?

5 Are you aware of any indexing
6 system used over there?

7 A I do not know.

8 Q You've never made reference to documents at
9 headquarter's office of the Medical Director?

10 A Yes.

11 Q Did you use an index system to locate the
12 documents you're looking for?

13 A I would ask the secretaries to retrieve them for me.
14 I do not know where they -- how they located
15 them.

16 Q Okay. You don't know if there was some sort of
17 catalogue or index or a table of contents of some
18 sort?

19 A I don't know.

20 Q All right.

21 Have you personally had any
22 involvement with computer retrieval of information
23 since 1977 --

24 A No.

1 Q -- relating to PCBs?

2 A No.

3 Q Okay.

4 MR. SCOTT: He may be able to
5 answer your first question then.

6 I'll talk with him after the
7 deposition and then maybe we can answer your
8 question on the record.

9 MR. LEDBETTER: Okay. And then
10 just get it all taken care of.

11 BY MR. LEDBETTER:

12 Q Is the library kept at the Medical Director's
13 office referred to as the Environmental Health
14 Library?

15 A I do not know how it's referred to.

16 Q Have you ever heard that expression; Monsanto's
17 Environmental Health Library?

18 A No.

19 Q Have you ever heard of a program referred to as
20 Monsanto's Six Point Program as it relates to
21 PCBs?

22 A Yes.

23 Q What is the Six Point Program?

24 A This program was approved by the corporate

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1 administration committee in early 1970, and it
2 had six elements to it. I'll try to recall them.

3 One had to do with continuing the
4 toxicology testing of PCBs.

5 Another element had to do with the
6 continue to refine the analytical methodology.

7 The third element: Withdraw from
8 those applications that were difficult to control
9 and led to easy escape to the environment.

10 Q That's number four?

11 A These are not in any specific order.

12 Q Control the escaping of PCBs in the environment was
13 the fourth one?

14 A By limiting the marketing to those applications.

15 Continue to communicate to
16 customers and the regulatory agencies any new
17 developments Monsanto became aware of.

18 What was the sixth point? It
19 escapes me at the moment.

20 I can't recall the sixth item.

21 Q Was there something about, for example, disposal?

22 A Oh, yes. Look for a technology that would assist
23 in the disposal of waste materials. And coupled
24 with that was the control of our operations

1 in such a way that losses down the sewer and up the
2 vent stacks would be controlled to some acceptable
3 level.

4 Q All right. Do you recall sometime in the early
5 '70s, that the recommended disposal practices
6 of PCBs changed at Monsanto?

7 A Yes.

8 Q What was the change, and what approximately was
9 the time period of that change?

10 A I mentioned early 1970. It was sometime in the
11 summer of 1970 that we were recommending that the
12 preferred disposal of liquid scrap material,
13 unusable, would be by incineration under the right
14 conditions.

15 Q And incineration was what we touched on earlier;
16 about it being at the right temperature to turn
17 the PCB product into essentially two gases?

18 A Really three.

19 Q There was hydrochloride and then there was carbon
20 dioxide?

21 A And water.

22 Q And water. And that had to be done at the right
23 temperature?

24 A Yes.

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1 Q Was Monsanto doing that at that time period; the
2 summer of 1970?

3 A No, but we had demonstrated by the summer of 1970
4 that it was doable.

5 Q Was it Monsanto that demonstrated it?

6 A No. It was the John Zink Company of Tulsa,
7 Oklahoma, that was in the petroleum furnace business
8 designing high temperature furnaces, that demonstrated
9 they could design a unit to handle PCBs.

10 Q Okay. And then that would fully complete the
11 environmental process so that it's just disposed
12 of harmlessly?

13 A Yes.

14 Q When did Monsanto start doing that?

15 A The unit was installed in 1971, and placed in
16 service about the summer of '71.

17 The material for disposal was collected
18 starting in 1970. So we had an inventory of material
19 that had been accumulating for about a year before we
20 started destroying it.

21 Q Is it fair that as early as 1969, Monsanto undertook
22 a program as part of that Six Point Program, to
23 develop a process for incineration?

24 A Oh, yes.

1 Q And eventually it was put into use in 1971, in one
2 location?
3 A Yes.
4 Q And eventually multiple locations?
5 A No.
6 Q Just one?
7 A Just one by Monsanto.
8 Q Okay. Customers used their own individual
9 facilities, do you recall?
10 A One customer installed their own unit. Some customers
11 used commercial units that were becoming available in
12 71, '72.
13 Q Did Monsanto provide recommendations or communications
14 to its customers to notify them to return the spent
15 fluids to Monsanto for disposal?
16 A Yes.
17 Q Was that started again in the 1970 period where
18 the collection began?
19 A Yes.
20 Q Were customers told why Monsanto wanted the material
21 to be returned to Monsanto rather than them disposing of
22 it themselves?
23 A Yes. We had told them to not dispose them in
24 such a way that it gets in the environment.

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1 And in response to their question,
2 what's the best way, we said we now know we can
3 incinerate.

4 You can send your material to
5 us at a given location for a given fee, and we
6 will do it for you.

7 Q What was the recommended disposal practice prior
8 to the incineration?

9 A Authorized landfills.

10 Q In containers, drums, and that sort of thing, in a
11 landfill?

12 A Yes.

13 Q Were there studies conducted by Monsanto
14 particularly relating to replacement chemicals
15 for dielectrics?

16 A Yes.

17 Q When were those started?

18 A The early work started in about 1969.

19 Q And this is all about that same time period that
20 this environmental thing came to light, right?

21 A Yes.

22 Q Was it found that there were suitable
23 replacements for dielectric use?

24 A Monsanto did not succeed in finding a suitable

1 replacement. So, they abandoned the market
2 eventually.

3 Q Does that mean transformers didn't get any fluid
4 anymore and capacitors didn't get fluid
5 anymore?

6 A The alternative materials, as I understand it,
7 did not have the fire resistance that the PCB fluids
8 had. So, the trade-off had to take place.

9 And Monsanto's attempt to find a
10 fire resistant fluid which was also a good
11 dielectric was unsuccessful.

12 Q Okay. And that would include that group of
13 dielectrics that you described for me; 1242,
14 1254, 1260? You couldn't find a suitable
15 replacement for those?

16 A That is right.

17 Q As I understand it, the dielectric fluid
18 production ceased in 1977?

19 A Yes.

20 Q Prior to that time, did the Aroclor 1260 cease
21 production?

22 A Yes.

23 Q And when was that?

24 A 1970.

1 Q Okay. Why was Aroclor 1260 stopped before the
2 others?

3 A It represented the highest chlorinated liquid,
4 and there was a very easy replacement in the
5 1254.

6 And the customers were very
7 willing to make that replacement. It apparently
8 made no difference in their final product.

9 Q Do you recall in terms of production, say, in
10 the five years between 1965 and 1970 that you
11 were at the Krummrich plant, in terms of
12 production, how the amount of PCB compared --
13 I'm sorry, Aroclor 1260 compared to Aroclor 1254
14 or 1242?

15 A It was the smallest of the three.

16 Q So, it was less of a problem to replace, I
17 presume?

18 A Yes.

19 Q Was there any conclusions, to your knowledge,
20 reached that since 1260 seemed to be a greater
21 burden on the environment, that that would be the
22 reason for ceasing production in 1971?

23 A In 1970, yes. That was the reason.

24 Q Okay. And 1254, you've indicated to us earlier,

1 was slightly less chlorinated, but still found in
2 the environment, but less of a long-lasting
3 effect than 1260?

4 A Relatively speaking, yes.

5 Q Okay. Do you know if liquid transformers have
6 been manufactured since 1977?

7 A I am told so, yes.

8 Q And no longer containing PCB products of any kind?

9 A That's correct.

10 Q Do you know what they've been replaced with?

11 MR. SCOTT: Isn't this a question
12 for GE?

13 MR. LEDBETTER: Well, I guess it is,
14 kind of.

15 MR. SCOTT: Why don't you ask him
16 questions about his area and let Dick's witness
17 answer the other questions.

18 BY MR. LEDBETTER:

19 Q Do you know what they've been replaced with?

20 MR. DINON: Foundation.

21 THE WITNESS: I believe I do, yes.

22 BY MR. LEDBETTER:

23 Q Okay. And what were they?

24 A Some were replaced with mineral oil and

1 installed in a different kind of environment.

2 Some were replaced by so-called
3 dry units.

4 Some are replaced by silicone
5 fluids.

6 Q And in Monsanto's opinion, none of those were
7 suitable prior to 1977 as replacements?

8 A They were not, in Monsanto's viewpoint, a like for
9 like kind of replacement.

10 The installation of these units
11 is different from what they would have been if
12 they had been PCB units. Sprinkler systems had to
13 be designed, different vaults --

14 Q Size also?

15 A Size. Or they had to forego fire resistance,
16 pay high insurance premiums, put in firewalls.
17 The conditions are different.

18 Q Okay. Did you do studies -- not you personally,
19 but Monsanto do studies in this '65 through '77
20 period, on biodegradability of PCBs?

21 A Yes.

22 Q And what studies specifically were those, if you
23 recall?

24 A They were two active programs. One in our

1 facility in Wales, United Kingdom. The other in the
2 St. Louis Research Center. They involved the
3 exposure of PCBs to bacteria and the
4 determination of what effect these bacteria
5 had under conditions of air flow and
6 concentration. The intent was to determine
7 if any of the PCBs were destroyed by these
8 bacteria.

9 Q And the findings?

10 A The findings are that the lower-chlorinated
11 biphenyls seemed to degrade fairly rapidly. Not
12 as fast as sugar water, but faster than many
13 chemicals.

14 The higher-chlorinated biphenyls
15 resist degradation. And the indication is that
16 they would last for many years in the
17 environment.

18 Q And the higher-chlorinated levels, that's 1254
19 and 1260 and one other you had mentioned
20 earlier on?

21 A There are components in those commercial mixtures
22 that reflect the higher. That is, the 5 chlorine,
23 6 chlorine, 7 chlorine, and higher, are very
24 resistant. And they are key components in the

1 Aroclor 1254 and 1260.

2 Q Do you recall who it was or what group in St.
3 Louis did the studies involving degradability?

4 A It was a group under the direction of Dr. Keller
5 through Dr. Scott Tucker. And I remember a Dr.
6 Vic Saeger being one of the researchers.

7 Q S-a-e-g-e-r?

8 A S-a-e-g-e-r. And then I believe there's a Dr. Emery
9 who was involved as well, in the early stages of
10 these studies. E-m-e-r-y.

11 Q To your knowledge, was that study confined to
12 the biodegradability question only, done by Dr.
13 Keller through Dr. Tucker and Dr. Saeger?

14 A Yes.

15 Q I just have a couple more questions for you and
16 then we're all done; at least I'm all done.

17 Mr. Papageorge, do you recall if the
18 Standard Operating Practices Manual had a warning
19 something to this effect, regarding chlorine gas,
20 saying: If you are caught without a gas mask and
21 large amounts of chlorine gas are liberated,
22 hold your breath and go cross-wind until you're
23 out of range of the gas. Once out of the area,
24 obtain emergency gas mask and return to the

1 area to correct the problem?

2 A Yes.

3 Q And the chlorine gas was used as a part of the
4 reproduction of PCBs -- I'm sorry, the
5 production of PCBs, correct?

6 A Yes.

7 Q Is chlorine or chlorine gas an accelerator of
8 toxic effects of the biphenyls?

9 A I'm not accustomed to that kind of terminology.

10 Q I'm just trying to describe in summary form, the
11 effect that we discussed earlier as to how
12 biphenyls are potentially toxic, and then how
13 they're potentially toxic after chlorine is
14 added.

15 It increases the toxicity, in
16 general?

17 A Yes.

18 Q Okay. Do you recall if the Standard Operating
19 Practices Manual may have included a section
20 relating to Aroclors which stated: "Other hazards
21 of the material: This material causes eye, skin
22 and mucous membrane irritation"?

23 A (Nodding affirmatively.)

24 Q Do you recall when you first read a Standard

1 Operator's Instructions or Procedures Manual
2 of this type?

3 MR. SCOTT: You've got jumbled up
4 about three questions.

5 What's your question to the
6 witness?

7 MR. LEDBETTER: When he first read
8 a Standard Operating Procedures Manual of this
9 type.

10 MR. SCOTT: Of what type?

11 MR. LEDBETTER: That contains these
12 particular warnings.

13 MR. SCOTT: Well, you haven't even
14 established that they did contain the last
15 warning that you just read.

16 MR. LEDBETTER: I think he just
17 answered that.

18 THE REPORTER: He nodded his head
19 affirmatively.

20 MR. SCOTT: Oh, all right. I'm sorry.

21 BY MR. LEDBETTER:

22 Q Let's go back to your response. You've got to
23 give me a yes or no response to the last
24 question.

1 And that was: Do you recall if
2 the Standard Operating Practices Manual contained
3 a section that said under Other Hazards: "This
4 material causes eye, skin and mucous membrane
5 irritation"?

6 A I do.

7 Q And when was the first time that you remember
8 reading a Standard Operating Practices Manual
9 pertaining to PCBs that contained such a
10 statement?

11 A About 19 -- well, let me look here and refresh
12 my memory.

13 I was going to say 1962, '63.

14 Q Okay. And I believe from our discussions earlier,
15 that you thought these Standard Operating
16 Practices Manuals may have gone back into the
17 '50s?

18 A Yes.

19 Q Okay. Also, do you recall a statement in the
20 Standard Operating Practices Manual pertaining to
21 PCBs, which indicated under other hazards: "This
22 material can cause dermatitis, systemic poisoning
23 from the fumes, yellow atrophy of the liver"?

24 A I remember. Yes, I do.

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1 Q Okay. Do you know what is meant by systemic
2 poisoning from the fumes?
3 A As a layman, that tells me that it's going to
4 poison my system. I'd better not get exposed
5 to it.
6 Q Okay. And what is yellow atrophy of the liver?
7 A I understand that that's a deposit or a buildup
8 of fatty material in the liver.
9 Q Is that an explanation for the larger size livers
10 that have been shown in the studies?
11 A That is one explanation that I have seen.
12 Q Also under other hazards relating to the
13 Aroclors: "There are skin, mucous membranes and
14 eye hazards encountered in the handling of this
15 material."

16 Do you recall that?

17 A Yes.
18 Q Then under the storage and handling, do you
19 recall the section that says: "Protect the skin,
20 lungs and eyes by wearing approved protective
21 clothing, respirator and goggles. People who
22 handle this material at elevated temperatures should have
23 medical supervision and the air should be
24 sampled to ensure compliance with MAC"?

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1 A Yes.

2 Q What is MAC?

3 A Maximum allowable concentration.

4 Q Okay. And that was a little different standard

5 than that suggested by the industrial hygienist,

6 which was a threshold value, right?

7 A Well, it meant the same thing. As far as

8 Monsanto was concerned, that was the maximum

9 allowable.

10 Q Do you have an understanding as to what is meant

11 by elevated temperatures?

12 A The intent there was to avoid -- it's other than room

13 temperature; or ambient temperatures.

14 Q Okay. And then in those situations, one should

15 have medical supervision and the air should be

16 sampled periodically?

17 A Yes.

18 Q And then under Aroclors, do you recall a section

19 that said: "Aroclors is manufactured in

20 Department 246, or non-flammable liquids, which may

21 vary greatly in viscosity in room temperature.

22 Contact with skin should be avoided. In case

23 of spills on the body, wash the affected area

24 with soap and water. Inhalation of Aroclor and

1 Montar fumes should be avoided as they are highly
2 toxic. Protect the skin, lungs and eyes by
3 wearing protective clothing, respirator and
4 goggles. If spilled on clothing, change to clean
5 clothes"?

6 MR. SCOTT: Counsel, you're now
7 reading from a document.

8 I request that you let the witness
9 take a look at it.

10 MR. LEDBETTER: Sure.

11 MR. SCOTT: Let's mark it and make
12 it an exhibit.

13 (Whereupon Deposition Exhibit 10
14 marked by reporter)

15 BY MR. LEDBETTER:

16 Q Here's the part that I was reading from. (indicating)

17 MR. SCOTT: Let the record
18 indicate that we've had marked as Deposition
19 Exhibit 10, what appear to be portions from a
20 deposition of Mr. William B. - oh, no, testimony
21 given by Mr. Papageorge in Bad Axe, Michigan;
22 excuse me. Selected excerpts of testimony.

23 MR. LEDBETTER: Okay.
24

1 BY MR. LEDBETTER:

2 Q First, Mr. Papageorge, would you take a look at
3 that and just see if, to the best of your
4 recollection, that rings a bell as to your trial
5 testimony in Bad Axe, or a portion thereof?

6 MR. SCOTT: A very small portion
7 thereof.

8 THE WITNESS: It appears to be an
9 excerpt of my testimony, as best I can tell.

10 MR. LEDBETTER: I've only got two
11 more paragraphs, and we're done.

12 BY MR. LEDBETTER:

13 Q Then do you recall a section in the Standard
14 Operating Practices Manual that indicates:
15 "The operating instructions are the property of
16 Monsanto Industrial Chemical Companies, and the
17 recipient is responsible for its safekeeping
18 and contains confidential information of
19 Monsanto"?

20 A Yes.

21 Q And under, again, other hazards -- I think we've
22 talked about that. As to the safehandling of
23 the material, do you recall the section: "A
24 clean change of clothes is furnished daily to

1 each employee and time is allotted at the end of
2 each shift for bathing. In addition, one should
3 wash his hands and face before eating"?

4 A Yes.

5 Q And those were all recommended criteria for those
6 working in and around PCBs?

7 A Yes.

8 MR. LEDBETTER: I've got no other
9 questions.

10 Thank you.

11 MR. DECKER: I just have one I'd
12 like to clear up. It will take two seconds.

13 EXAMINATION

14 BY MR. DECKER:

15 Q Mr. Papageorge, my name is Bill Decker. I
16 introduced myself earlier, but that was quite a
17 while ago.

18 I represent Transformer Inspection
19 Retrofill who's involved in this case regarding
20 repairs of the subject transformers.

21 Just as a precedent, my knowledge
22 of chemistry, etcetera, compared to Mr. Ledbetter
23 is probably something like Einstein to Poindexter,
24 because I really have nothing.

222

1 So, I hope you'll help me in this
2 area to clear something up.

3 A I'll try.

4 Q You had testified earlier something concerning
5 the likelihood that PCB vapors would be present
6 if there was evidence of PCB running down the
7 side of a transformer and then collecting to some
8 degree on the ground.

9 Is that a fair recollection of
10 what you said?

11 A Yes, it is.

12 Q We also mentioned that in order to define what
13 kind of concentration we can estimate to have
14 in this room, there are other factors, such as
15 the size of the room, the heat, the ventilation
16 presence, etcetera.

17 That's also correct?

18 A That is correct.

19 Q Okay. Would it be a fair assumption to say that
20 without having those measurements, including the
21 heat involved in the room, would there be any way
22 of determining the concentration of PCB vapors in
23 such a room?

24 A At the time the --

223

1 Q Alleged exposure occurred.

2 A At the time, yes, there are ways to do this.

3 Q Okay. I will get to that in one second.

4 At room temperature, if the PCB
5 material had collected on the floor, are the
6 molecules light enough that they would ascend? Or
7 would they stay close to the floor?

8 Or are there other factors
9 involved in order to make that determination?

10 A In every liquid, there are always some molecules
11 that are going to escape. The energy with which
12 they escape varies from liquid to liquid.

13 PCBs would lose very few molecules
14 compared to the benzenes that we talked about
15 earlier.

16 Q Okay.

17 A With today's very sensitive instruments, the few
18 that do escape would be detected. Then it
19 becomes a matter of: Are there enough to cause
20 harm?

21 Q Okay. We had discussed earlier the amounts that
22 Monsanto had -- or the parameters that we were
23 working with before; the one milligram and one
24 and-a-half milligram, depending on which Aroclor we

1 were working with?

2 A That is correct.

3 Q Okay. Would those instruments, those very
4 sensitive instruments, be able to make that
5 determination?

6 A Easily. Yes.

7 Q Okay.

8 MR. DECKER: Okay. I have no
9 further questions.

10 MR. SCOTT: Okay. Let me ask him one
11 question off the record.

12 (Off the record)

13 MR. SCOTT: I'm not going to let
14 him answer the question. You can submit a
15 special interrogatory to me if you want, and we'll
16 deal with it.

17 MR. LEDBETTER: Okay.

18 (At about 5:05 p.m. - whereupon
19 deposition concluded)

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CERTIFICATE OF DEPOSITION REPORTER

STATE OF MICHIGAN)

COUNTY OF ST. LOUIS)

I, Lori B. Resnick, Notary Public in and
for the County of Wayne, State of Michigan, do hereby
certify that the foregoing deposition of WILLIAM B.
PAPAGEORGE, was taken before me at the time and place
hereinbefore set forth, and that said witness was duly
sworn by me to tell the truth, the whole truth and
nothing but the truth, and thereupon was examined and
testified as in the foregoing deposition appears;

That this deposition was taken and
thereafter transcribed by me and that it is a true
and full report of my original stenomask notes.

IN WITNESS WHEREOF, I have hereunto
set my hand this 27th day of June, 1987.



LORI B. RESNICK, CSMR 3236
Notary Public, Wayne County, MI
Acting in
My Commission expires: 12/29/87

23/24 men in manufacture of chlorinated biphenyl
developed chlor-ace.
problem seemed to be worse with manufacture of
a poor grade of chlorinated biphenyl.
poor hygiene at plant.

Arch. Derm.
Syph.
33:1022, 1936

AN ACNEFORM DERMATERGOSIS

JACK W. JONES, M.D.

AND

HERBERT S. ALDEN, M.D.

ATLANTA, GA.

It is only within comparatively recent times that the dermatologist has interested himself in dermatoses associated with or caused by the daily work. With the advent of a more clearly defined appraisal of the words eczema and dermatitis and the more general use of the patch test, dermatologists have necessarily inquired more and more deeply into the innumerable chemical contacts that occur in the day by day lives of their patients. As well as being conversant with dermatology, the dermatologist must have a working knowledge of the manufacture and content of the many new and old compounds that are handled and applied by large numbers of persons. Hence, more and more dermatologists are becoming industrially minded, and it is dawning on them that the solution of some of their industrial difficulties may aid them in a clearer conception of the dermatoses that are encountered in their every-day practice.

Dermatargoses of the acneform type occurring in persons working with chemicals have been repeatedly reported in Europe and discussed as industrial "chlor-ace."¹ This term "chlor-ace" was first used by Herxheimer² in 1899 to describe an eruption composed of comedones and small sebaceous pustules that occurred on the arms and faces of workers manufacturing chlorine gas electrolytically, using carbon electrodes. It was natural to assume that the chlorine was the causative agent. Bettman³ had observed in March 1897 two patients whose skins were diffusely pigmented, dark, rough and dry, in whom were observed small tenacious comedones associated with numerous small follicular abscesses. Subsequently he reported twenty-one additional cases with similar symptoms in workmen who were engaged in cleaning out an acid tower used in the manufacture of hydrochloric acid. At no time did these men come in contact with free chlorine, but numerous loose

CYNTHIA M. WEICHRMAN

Carol L. Davis

Reporting Service

Houston, Texas

Exhibit 14

Read at the Fifty-Eighth Annual Meeting of the American Dermatological Association, Inc., White Sulphur Springs, W. Va., May 2, 1935

1. White, Prosser: The Dermatargoses, ed. 4, New York, Paul B. Hoeber, Inc., 1934.

2. Herxheimer: Munchen. med. Wchnschr. 46:278, 1899.

3. Bettman: Deutsche med. Wchnschr. 27:437, 1901.

DEPOSITION
EXHIBIT

#1 5/27/87
L.R.

derivatives of coal tar were present. Herzheimer later absolved the chlorine but considered the trouble to be due to various chlorbenzenes.

From time to time other reports of the occurrence of "chlor-acne" have been made in the European journals, each author continuing the error of Herzheimer in assuming that the cause of the condition was chlorine. Some of these reports have been indefinite as to the chemical conditions under which the men worked, except that they were exposed to chlorine gas. However, some authors have suspected various chlorbenzene derivatives, such as hexa-chlorbenzene, hexa-chlorethylene, para-nitrobenzene, perchloronaphthalene ("perna") and probably others.⁴ Chlorinated naphthalene was first indicted by Wauer⁵ in 1918 as a cause of acneform eruptions of the skin, the condition being called *Perna-krankheit*. His observations were confirmed by Koelsch⁶ and recently by Teleky.⁷ The latter reported an exhaustive study of a large number of cases occurring in workers engaged in the manufacture of perchloronaphthalene, in which the workers were exposed to fumes of the molten mass and also to the sublimated dusts. In approximately one-half of the workers exposed there developed comedones of a particularly heavy and tenacious type with resultant sebaceous abscesses, containing heavy yellow pus and serum. Considerable improvement resulted when the chlorine content of the mass was decreased from 30 per cent to 8 per cent, and Teleky believed that the disease was due directly to the halogen content of the substance. He mentioned the similarity of the disease to *tearacne* (tar acne) but carried the analogy no further. Individual predisposition appeared to alter receptivity.

Some authors have leaned to the view that the acneform eruption is due to direct external contact with the chemicals (notably Bettman, Hallopeau, Jacquet, Fumouze, Teleky and Koelsch⁸). Others (Lehmann, Herzheimer, Jacobi, Roth and Kolert⁹) considered it to be a

4. Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1925, brochures 34 and 285.

5. Wauer, cited in Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1925, brochure 34.

6. Koelsch, F., in Ullmann, K.; Oppenheim, M., and Rille, J. H.: Injuries to the Skin, Leipzig, Leopold Voss, 1926, vol. 2, p. 303.

7. Teleky: Klin. Wchnschr. 6:845 (April 30): 897 (May 7): 1927, 7:214 (Jan. 29): 1928.

8. Bettman; Hallopeau; Jacquet; Fumouze; Teleky, and Koelsch, cited in Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1925, brochure 285.

9. Lehmann; Herzheimer; Jacobi; Roth, and Kolert, cited in Occupation and Health: Encyclopedia of Hygiene, Pathology and Social Welfare, International Labour Office, Boston, World Peace Foundation, 1925, brochure 285.

dermatitis due to the absorption of chemical compounds by the lungs or gastro-intestinal tract, with elimination by way of the sebaceous glands. The majority of writers on the subject, particularly Prosser White,¹ have seemed to feel that chlorine as such has little to do with the formation of the comedones and cysts, and they have repeatedly referred to tar and products of the distillation of tar as the prime causative factors. Prosser White rebuked authors for using the term "chlor-acne" at all and expressed the belief that the process is one of the manifold cutaneous reactions produced by tar and its derivatives.

Recently we have had the opportunity to study, with the full cooperation of the manufacturers, an outbreak of acneform eruption occurring in a group of workers engaged in the manufacture of chlorinated di-phenyl. This study has brought out some points in the production of this unusual dermatosis that we feel are important in solving its exact cause. The following case is typically illustrative of the disease as it occurred in the men working in this plant.

REPORT OF A CASE

History.—O. D., a Negro aged 26, began work in the distillation of chlorinated di-phenyl in April 1933 and worked regularly until the latter part of the year 1933. About May of 1933, he noticed the appearance of blackheads on his face, neck, arms and legs. These areas itched slightly. In a short time blackheads began to appear on the chest, back and lower part of the abdomen, around the navel and on the scrotum and penis. Many of these blackheads swelled and became infected, discharging thick pus. The areas healed with difficulty and often left scars. The condition seemed to be progressive until November 1933. When seen in December 1933 the patient complained of lassitude, loss of appetite and loss of libido and said that his cutaneous condition seemed to be improving.

Physical Examination.—On examination he seemed in good general health. His complaint of lassitude was not borne out by anything more than the usual temperament of the Negro toward work. On the forehead, extending within the hair line, and on the cheeks, chin, nose and neck were numerous small, very black, tenacious comedones, their distribution best described as being "peppered" within the skin. Many of the comedones surmounted firm shotlike cysts, which in some areas contained viscid yellow pus. The pustular elements were more noticeable on the neck. Similar shotlike comedones and cysts had appeared on the shoulders, midportion of the back and chest, with an occasional large cyst. A peculiar peppering of the skin with tenacious carbon-colored comedones was apparent around the umbilicus and lower portion of the abdomen. The scrotum and penis were involved in a similar process, the former being given more to the formation of cysts. The outer surfaces of the forearms and anterior thighs showed similar but fewer comedones. The whole eruption was acneform but differed from acne particularly in the lack of a seborrheic appearance of the skin and in the peculiarly deep black of the comedones as well as the general peppered distribution in areas not usually involved in acne vulgaris. A general physical examination had revealed nothing of importance.

Treatment.—He was instructed to scrub his skin thoroughly before and after his hours of work, to wear fresh clothing each day during work and to use a lotion made up as follows: zinc sulfate 4 Gm., potassium sulfurate 4 Gm. and distilled water to make 125 cc. On recommendation he later reported for weekly doses of roentgen radiation according to the usual manner of treating acne vulgaris.

Microscopic Examination.—A portion of the skin of the chest was removed for microscopic study (fig. 1). The chief feature of the sections was noted in the hair follicles and sebaceous glands, in which there were cystic dilatation,

Summary of the Symptoms and Treatment in Sixteen Cases of Acneform Eruption

Case	Age	Race	Type of Skin	Type of Eruption	Time of Exposure	Special Treatment
1H	22	White	Solorrhetic; previous acne	Diffuse comedones; few cysts	6 months	Incision; roentgenotherapy
2B	25	White	Average	Diffuse comedones; few cysts	5 months	None
3S	22	White	Solorrhetic	Diffuse comedones; large cysts and pustules	Throughout	Incision; drainage
4W	6	White	Average-dry	Diffuse comedones; large cysts and abscesses	10 months	None
5F	25	White	Average	Diffuse comedones; few cysts on face and neck	Throughout	None
6S	9	White	Solorrhetic; previous acne	Diffuse comedones; deep abscesses on neck; severe cysts	10 months	Incision; drainage
7B	20	Negro	Solorrhetic	Erythematous diffuse comedones; few small cysts	6 months	None
8B	9	Negro	Average	Few scattered comedones; occasional cyst	5 months	None
9C	2	Negro	Solorrhetic	Diffuse comedones; cysts; small abscesses	Throughout	Roentgenotherapy
10C	7	White	Average	Scattered comedones; occasional cysts	6 months	None
11C	5	White	Dry	Scattered comedones; occasional cysts	Throughout	Roentgenotherapy
12P	20	White	Solorrhetic	Few comedones; occasional cysts	2 months	None
13H	7	White	Average	Occasional comedones	Throughout	None
14B	21	Negro	Average	Very few comedones	12 months	None
15F	22	White	Solorrhetic	Scattered comedones	Throughout	Roentgenotherapy
16P	20	Negro	Solorrhetic	Diffuse comedones; few cysts back and face	Throughout	Roentgenotherapy

* This case is reported in detail.

destruction of the hair, marked thinning and atrophy of the epithelium of the follicles and a heavy plug of keratinized material which partly filled the cystic cavity. In some areas there was a superficial plug at the surface opening; others showed the surface open and the plug deeply situated. There was no purulent exudate. There was a zone of moderately dense connective tissue surrounding the enlarged follicles, slight edema and infiltration by lymphocytes but no leukocytes. Slight edematous changes were noted in the occasional sebaceous glands present, but they were strikingly few. The sweat glands were normal.



Fig. 1.—Section of skin from the chest, showing histologic changes in the formation of an acneiform eruption.

Of the twenty-four men working in the manufacture of chlorinated di-phenyl within the period from the late summer of 1932 to October, 1933, twenty-three were reported to have had an acneform eruption on the face and body. Of the twenty-three, sixteen were examined. These men presented eruptions of acneform character similar in type and distribution to that in the case referred to but varying in severity (table). In many patients numerous small sebaceous abscesses developed, particularly around the collar line, which exuded heavy, tenacious pus.



Fig. 2.—Characteristic appearance and location of an acneform eruption.

and the remaining ulcers were indolent, leaving in their wake much scarring. In two of the cases particularly, many large abscesses developed on the neck and back.

Until very recent times di-phenyl and chlorinated di-phenyl were only laboratory curiosities, and their commercial manufacture was unknown. In the experimental stage of manufacture the apparatus was necessarily crude, and experimenters and workers were exposed for long periods to fumes and dusts containing much chlorine, both free and combined. In the early experiments, as well as in the early manufacture in large

quantities, the di-phenyl was made by heating benzene derived from crude coal tar. When heated in a suitable medium benzene (C_6H_6) becomes di-phenyl ($C_{12}H_{10}$). This substance is then chlorinated at various saturation points, by exposing it under suitable conditions to free chlorine gas. Distillation of the resulting chlorinated di-phenyl¹⁰ results in a purified commercial product (fig. 3). The commercial benzene always has a small quantity of impurities, but some of the cruder commercial benzenes contain relatively large quantities of these impurities, such as xylene, toluene and paraffin. On the heating of the

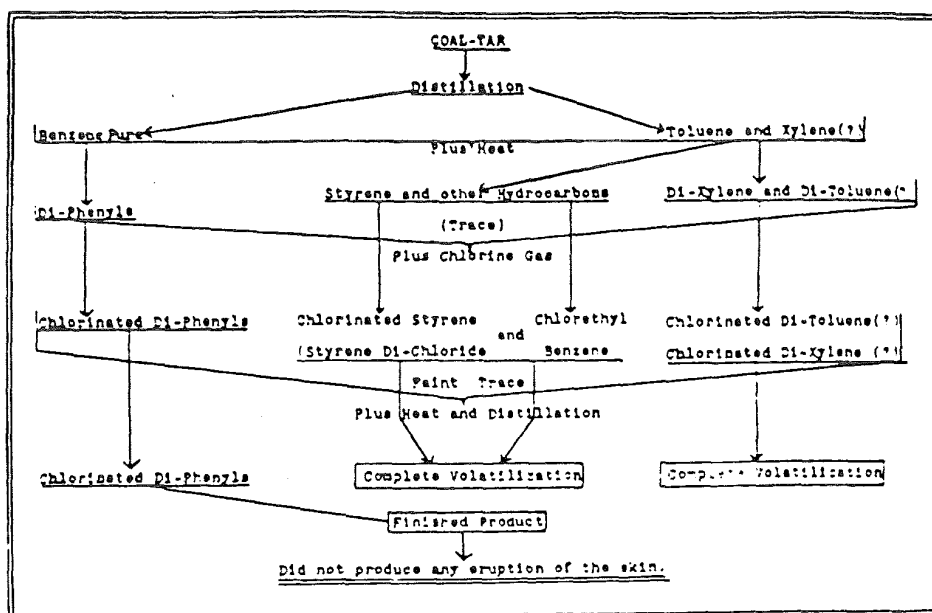


Fig. 3.—Chart illustrating the process of manufacture of chlorinated di-phenyl.

cruder benzenes, the toluenes and xylenes, when present in large quantities, may produce the chemical substance styrene, which has the formula $C_6H_5CH=CH_2$. Styrene when chlorinated (it being inseparable from the di-phenyl) is an unstable compound (ripping its chlorine atoms very readily). It is probable that other complex hydrocarbons may be formed from these impurities, which on chlorination become quite as unstable as the styrene. One of these compounds may be chlorethyl benzene ($C_6H_5CH_2CH_2Cl$).

10. Chlorinated di-phenyl consists of a mixture of di-phenyl chlorinated at various saturation points: nona-chloro-di-phenyl ($C_{12}HCl_9$), ($C_{12}Cl_9$) etc.

In the experimental stage and in the early manufacture of chlorinated di-phenyl, the men working were exposed for long periods to these chlorinated products. As the demand for the finished product increased, quantitative manufacture was speeded up rapidly, and open stills and heating units were of necessity used until better equipment could be designed and made. But since no physical trouble had developed in the workers previously there was no apparent hazard connected with manufacture. On or about March 1933 the electrical properties of the chlorinated di-phenyl produced in the plant the workmen of which were examined fell below the specifications, and the color of the product deepened. About one month later an acneform eruption was observed on the faces and arms of several of the workers, which became progressively worse, gradually becoming apparent among all the workers engaged in the process at that time. Although one of the men had had a slight acneform eruption on his face in January, little thought was given to it until the eruption appeared in the others. In the summer of 1932, six or eight months previous to the general outbreak of the condition, owing to a difference in price, the crude benzene was purchased from another source, and it was not until the purchase of this particular benzene was discontinued, in October 1933, that the finished product came up to the standard. During the period from March 1933 until October 1933, the eruption continued among the men, and it was present as already described when the men were examined in December of the same year.

The appearance of the eruption coincidentally with the production of a poor grade of chlorinated di-phenyl seemed to indicate a relationship. On blue chemical investigation styrene was assumed to be the offending substance causing the low grade of the finished product.

It seems that it may be readily assumed that our problem as to the causative agents of the acneform eruption was connected with the chlorinated products of the impurities present in the crude benzene. However, with the exception of styrene, these products are not known. The problem hence becomes difficult of exact solution, and it is necessary to resort to a reasonable deductive hypothesis.

The exact conditions under which the men contracted their eruption cannot be satisfactorily duplicated experimentally since prolonged mild contact seems to be a deciding factor. Repeated patch tests made on numerous persons with the finished product, chlorinated di-phenyl, and the foreign substances styrene, styrene di-chloride and chlorethylbenzene, have not resulted in an acneform eruption except in one instance. Repeated patch tests made with chlorethylbenzene in the same area produced on the third attempt an erythematous follicular eruption, which persisted only a few days but which was by no means typical.

We could not duplicate this in other instances. Since styrene, chlorinated styrene, chlorethylbenzene, xylene, toluene and their probable products are for the most part lipid solvents, they will produce death and denudation of the epithelium, and one always obtains a chemical burn in response to a patch test made with the styrene and styrene di-chloride. Small quantities mixed loosely with an inert powder or dissolved in ether produce a mild erythematous reaction but no acneform eruption. Patch tests made with the finished product, chlorinated di-phenyl, at no time produced any erythema or evidence of irritation; hence the chlorinated di-phenyl can be absolutely absolved as an irritating agent. In looking over the consecutive history of the development of the eruption, we found it apparent that the men were in some degree exposed to the chlorinated impurities in the benzene for some time before the eruption actually appeared, and that to duplicate the conditions we should have to make repeated patch tests daily for long periods in the same person, a procedure which is not feasible.

As a group, the workers in the dusts and vapors, which were presumably saturated with chlorinated hydrocarbons, were not cleanly, making little effort to bathe after work, and sweating and rubbing enhanced the liability to deposition of the substances on the skin. Theoretically, the chlorinated hydrocarbons, presumably styrene di-chloride and chlorethylbenzene, deposited on the skin gather around the hair follicles and sebaceous glands and are gradually rubbed in. Being lipid solvents, they dissolve sebum and in the reaction, which is modified by the heat of the body and the water present, give off hydrochloric acid, which causes death or irritation of the cells. This in turn causes an excess of cell growth, inflammation, sebaceous plugging and an acneform eruption (fig. 4). This process repeated over a long period may result in severe sebaceous infection, sebaceous abscess and scarring. One would naturally expect this process to occur in the seborrheic areas as well as where clothing might rub the particles into the skin. Also one would expect the process to enhance an already existing acne vulgaris. This occurred in cases 1 and 6 (table 1), the cases in which there was the worst involvement with sebaceous abscesses.

In the beginning an attempt at prevention of the condition was made by being especially careful that all men engaged in the manufacture of chlorinated di-phenyl should have a thorough bath after working hours and that they should wear freshly laundered clothing before starting work. They were also instructed to apply night and morning veterinary white lotion to the affected parts. Some of the patients having the most numerous cysts and follicular infections were instructed to report for roentgen treatment administered in the manner usually employed in treating acne vulgaris. Following the

change in the type of benzene used and the employment of enclosed distilling apparatus and ventilation fans, there was noted a gradual improvement in the acneform eruption. Those patients receiving roentgen radiation improved much more rapidly, but it cannot be said that this form of treatment gave unusual results. The final treatment, of course, was removal of the patient from the offending chlorinated hydrocarbons, and then cleanliness and relief from any infection. All the men showing evidence of cutaneous disease, so far as is known, are greatly improved, or the condition has entirely cleared except for an occasional comedo and a few scars.

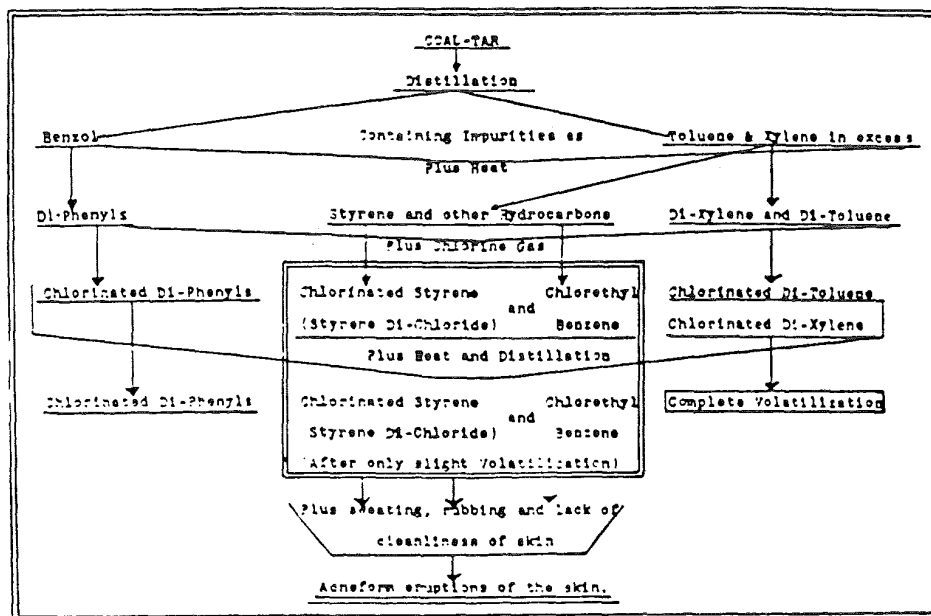


Fig. 4.—Process of manufacture of chlorinated di-phenyl containing impurities which cause an acneform eruption.

When one recalls the cases reported by the European investigators, it is altogether plausible that the chemical process outlined here occurred in most instances. The slow liberation of chlorine in the presence of a lipid solvent (usually chlorinated hydrocarbons) was present in most cases. It is doubtful whether the disease is influenced by the quantity of chlorine present; it is apparently more due to the formation of unstable chlorinated hydrocarbons as well as to the liability of the workers to close contact without a thorough cleansing of the skin and a change from saturated clothing. It is also probable that the disease has little to do with tar itself but more with the products formed by the

chlorination of tar. Hence, the disease cannot be called "chlor-acne" or "tar acne." That it is not caused by internal absorption is evident when one considers the following facts: First, acute intoxications by chlorine rarely occur, since suffocation and death by pulmonary edema ensue even in the presence of relatively small quantities of chlorine gas in the air. Second, when ingested, chlorine and the chlorinated hydrocarbons are so rapidly oxidized and chlorine so readily combines with sodium that opportunity for acne-like eruptions, such as occur in bromine intoxication, is not possible.

In retrospect, we are intrigued by the hypothesis that these theoretical considerations may have some relation to acne vulgaris. The past quarter of a century has seen an enormous increase in the use of coal for heating homes as well as in industry. The smoke stacks of the cities have belched an ever increasing stream of coal smoke—smoke heavily laden with carbonaceous products containing numerous hydrocarbons. It is well known that coal smoke contains hydrochloric acid. Is it not possible that at least some of the acne vulgaris may have a part of its origin in the constant contacts with the chlorinated hydrocarbons of coal smoke?

SUMMARY

We have recorded herein observations concerning an industrial dermatosis of an acneiform type. The type of eruption and the causative factors are analogous to those in the previously reported cases spoken of in the European literature as instances of "chlor-acne," tar acne or *Pernakrankheit*. Although exact duplication of the conditions under which the workers acquired the dermatosis cannot be accomplished by experimental means, reasonable deductions as to the cause of the difficulty can be made. The eruption occurred during the manufacture of chlorinated di-phenyl from benzene. The dermatosis occurred at a time when the benzene used contained excessive quantities of toluene, xylene and paraffin as impurities. The heating and chlorination of these impurities probably resulted in the production of styrene di-chloride and chlorethylbenzene, which, we believe, on contact with the skin produced the acneiform eruption by the slow liberation of hydrochloric acid and not by internal absorption of any chemical. While many chlorinated hydrocarbons, as brought out by Prosser White, are lipid substances producing death or irritation of the cells by the liberation of chlorine, this is not true in all instances, since the finished product, chlorinated di-phenyl, did not, either experimentally or actually, produce any cutaneous or sebaceous irritation. Irritation apparently depends on the ease with which chlorine atoms are released from the hydrocarbon base. We believe that these dermatoses should be labeled acneiform dermatergoses resulting from certain unstable chlorinated

hydrocarbons and should not be spoken of as "chlor-acne" or tar acne. The suggestion has been made that some of the acne vulgaris of the skin may be the result of the same type of irritation from soot or coal smoke.

ABSTRACT OF DISCUSSION

DR. OLIVER S. ORMSBY, Chicago: I should like to add an experience I had three months ago that is of interest in this connection. I think that Dr. Jones' paper is extraordinarily interesting and valuable, and the small group of cases I am going to report may add to his troubles rather than solve any of the problems.

About three months ago in Michigan, I saw a patient, through the courtesy of Dr. Milton G. Butler, who presented an extraordinary acneiform eruption. This patient was 18 years old and had been observed by Dr. Butler. The eruption in these various cases consisted of acneiform pustules and comedones of variable size and depth, with scars, some of which were keloidal. The eruption occurred on the face and neck, particularly over the posterior portion, and extended well up into the scalp. The trunk, arms, thighs and legs were also involved. Two of the patients presented on the back of the neck deep perifollicular pustular lesions, with keloidal scars, such as are seen in Kaposi's dermatitis papularis capillitii. Both deep and superficial atrophic scars, such as are seen in deep lesions leaving acne, were present in other portions of the neck, face and trunk. The comedones were prominent in all areas, including the legs. They were of variable size, and many were large and deep. The age of the affected patients varied from 18 to 50 years, the younger ones being more severely affected. The condition was apparently produced by contact with an ingredient in a fungicide produced by a chemical company for destroying fungi in lumber. The particular chemical in this fungicide that apparently was responsible for the eruption was sodium tetra-chloro-ortho-cresylphosphate, which is a yellow powder. Only those who came in contact with it had the eruption. Those who handled the other ingredients of the fungicide and did not come in contact with this particular chemical had no difficulty. As there were apparently no chlorine, bromine or tar radicals in the formula, it is difficult to determine the active factor in the production of the lesions and also whether the eruption was caused by local action by contact or through absorption and internal effects. The eruption was not concerned in any way with acne, and no patient in whom the eruption developed had had acne previously.

DR. MARION B. SULZBERGER, New York: I think that Dr. Jones has contributed a study that will be very interesting when continued. I had an opportunity of seeing a group of patients with acne of external origin about a year and a half ago. The patients were all employed in a factory making radio condensers. Pitch tar and a type of wax (halowax, a chlorinated naphthalene) were used in these condensers. The acne which developed was like that described by Dr. Jones and also included comedones on the legs, as did that which Dr. Ormsby has mentioned. I saw six patients from this one small factory. They were all in the age of puberty or the early twenties, and the group included persons of both sexes.

I believe the most important point in this study is the possible connection between acneiform dermatoses distinctly due to external irritants and true acne vulgaris. It is interesting that many derivatives of tar are closely allied to the estrogenic hormone, that certain components of tar are both estrogenic and acnegenic and that the estrogenic hormone must perhaps be incriminated in the production of acne vulgaris. There can be no doubt that acne is often due to stimulation or

irritation of the pilosebaceous apparatus and that hormonal as well as external influences can produce this stimulation. In the patient suffering from acne, who has, of course, come in contact with the estrogenic hormone during intra-uterine life and then has had no further contact with this hormone (or the closely related testicular hormone) until he or she begins to form his or her own hormones, one can readily conceive the pathogenesis of acne vulgaris as being based on an excessive stimulation of the hair follicles, a stimulation produced by the sudden new formation of the hormones at puberty. It is as though the follicles, after being sensitized to the hormones during fetal contact and then passing through a period of freedom from contact, react excessively to the new and massive contacts with the products of the gonads at puberty. I have discussed this subject in great detail elsewhere.

DR. H. G. IRVINE, Minneapolis: I wish to comment on one point and to compliment Dr. Jones on an excellent piece of work and his presentation of it. There was a difference between his situation and that which Dr. Tunnacliff and I encountered (ARCH. DERMAT. & SYPH. 33:306 [Feb] 1936). In his case the manufacturers called on the medical profession to help solve the problem. Our study was undertaken where lay people were in charge. It shows clearly the need of medical advice in the production of various chemical preparations.

DR. JACK W. JONES, Atlanta, Ga.: I am very grateful for the discussion. I did not have time to go into the various preventive measures attempted in controlling the outbreak or the treatment. In most of the cases the condition has finally cleared up.

Regarding Dr. Sulzberger's discussion, we studied this outbreak and the literature rather carefully for a year and a half or two years, and we are convinced that the etiology in this particular group can be ascribed to external irritation and not to any internal complications. Our idea is that the unstable chlorinated hydrocarbons give off chlorine very easily, whereas the chlorine that is fixed in the benzene ring is comparatively stable. When these unstable chlorinated hydrocarbons come into contact with the skin and enter the hair follicles, they are broken down slowly, giving off chlorine to form hydrochloric acid. We feel that this is the primary cause of the trouble.

Dermatitis from Synthetic Resins and Waxes*

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DERMATITIS in the manufacture of natural resins and from their use has been reported by numerous observers,¹ but dermatitis in the manufacture of synthetic resins and from their use has not been frequently reported² or studied.

The principal synthetic resins manufactured in the United States are:

1. Phenol-formaldehyde resins, sold under various trade names, such as Bakelite, Durite, Durez, Resinox, Indur, etc.
2. Urea-formaldehyde resins, sold under trade names such as Beetle, Plaskon, Unite
3. Coumaron resin, made from coal tar residue
4. Ester gums, which are natural resins, such as Rosin, Dammar and Copal, in which the acid of the resin has been combined and neutralized with glycerol
5. The glyptal resins, which are a combination of glycerol and an organic acid, such as phthalic and malleic acids
6. The vinyl resins, which are vinyl esters, ethers or halides combined with phenol, formaldehyde and hexamethylenetetramine
7. Furfural resins, obtained from combining furfural with phenol (furfural obtained from corn cobs by treating with H_2SO_4 and fractional distillation)
8. Chlorinated resins, obtained by chlorinating various oils and balsams
9. Chlorinated waxes, obtained by chlorinating mineral oils, paraffin, naphthalene, di-phenyl, etc.

These resins are used for many purposes:

*Read before the Industrial Hygiene Section of the American Public Health Association at the Sixty-fourth Annual Meeting in Milwaukee, Wis., October 8, 1935.

For making plastics, such as telephone receivers, pipe stems, push buttons, ear phones, cigar holders, bottle caps, buttons, inks, artificial teeth plates, ornaments, bracelets, earrings.

For making varnishes, flexible coatings, floor finishes.

For making cements, wall boards, gears, dishes, water containers, insulators, rubber compounds, and numerous other objects and for electrical insulations on condensers and wires.

The most important of the resins, from a standpoint of volume used and skin hazards, are the phenol-formaldehyde, the urea-formaldehyde and the coumaron resins.

PHENOL-FORMALDEHYDE RESINS

These are made in two principal varieties:

1. The cast resin, which is finished in the factory
2. The molding resin, which is sold to molding companies and completed in the molding process

Phenol or cresol, formaldehyde and ammonia are mixed in proper proportions in a kettle and heated a sufficient length of time and to the proper temperature.³ They combine and are drawn out of the kettle in the form of a syrup. This is run into pans and allowed to cool, when it solidifies. This is known as "first stage resin," or "alpha resin." The pans are then heated, the resin re-melted and poured into suitable molds. These are placed in large, mineral oil filled carriers and

placed into carriers are heated and beta and gamma completed cast resins.

After being the resin is tar oil, and washed and dried, and carved or lathed object is desired.

During the tions, formaldehyde of the room of it, unless air is employed.

who are sensitive develop dermatitis and arms, as parts where the belt line, and the wrist.

The oil use dissolves out phenol and formaldehyde over and over considerable quantities. On analyzed after contained 2% and formaldehyde this oil have it and at times acne and dermatitis and other parts soaked clothing tities due to the phenol and the in the oil. This is usually a condition on which there are pustules.

The girls resin, with so develop dermatitis arms from the strong soap hands are factories, the hydrate after neutralize the

placed into curing ovens, where they are heated and go through the so-called beta and gamma stages into the completed cast resin.

After being taken out of the oven, the resin is taken out of the molds and oil, and washed with soap and water and dried, and it is then ready to be carved or lathed or bored into whatever object is desired.

During the course of these operations, formaldehyde is given off and the air of the room is strong with the odor of it, unless adequate forced ventilation is employed. Workers in the rooms who are sensitive to formaldehyde may develop dermatitis of the face, neck, and arms, as well as of the covered parts where there is friction, such as the belt line, the ankle at the shoe top, and the wrist at the cuff line.

The oil used in the curing process dissolves out of the resin some of the phenol and formaldehyde and, if used over and over again, it can contain considerable quantities of these substances. One sample which was analyzed after it came out of the ovens contained 2 per cent each of phenol and formaldehyde. The men handling this oil have their clothes splashed with it and at times suffer not only from oil acne and dermatitis of the legs, thighs, and other parts touched by the oil-soaked clothing, but also from dermatitis due to the irritating action of the phenol and the formaldehyde contained in the oil. The eruption in these cases is usually a diffuse erythematous base on which there are scattered the papules and pustules of an oil folliculitis.

The girls washing the oil off the resin, with soap and water, may also develop dermatitis of the hands and arms from the oil and also from the strong soap solution with which their hands are constantly wet. In many factories, the oil is treated with sodium hydrate after each baking in order to neutralize the phenol and formaldehyde

it may contain, but if this is not carefully done, dermatitis may result from too much alkali.

The skin hazards to the users of these cast resins are practically nil, because when they are finished no phenol or formaldehyde are given off from them.

The molding resin is made up in numerous qualities, containing different proportions of phenol and formaldehyde and ammonia. It is carried to the alpha stage or beta stage, and then is ground, heated, and mixed with ground wood, zinc stearate, soap, dyes, and hexamethylenetetramine in a mix mill. From this mill it is discharged on to conveyors, where it is air cooled. While cooling, it forms lumps which are ground in mills to proper sizes fit for molding. The workers engaged in mixing, grinding, and packing the molding resin are all exposed to the dust of the irritating chemicals it contains, as well as to the formaldehyde which it gives off, the odor of which permeates the rooms. Dermatitis is a frequent occurrence in this occupation, especially if the rooms are not properly ventilated to draw off dust, and if the machines and jobs where there is dust are not properly protected by suction hoods.

In a factory employing about 400 workers, where no great care was taken to allay and prevent dust, 27 cases of dermatitis occurred in 8 months. Patch tests performed on 10 cases with various resins, hexa, 4 per cent solution of formaldehyde, and 2 per cent solution of phenol, showed that hypersensitivity to hexamethylenetetramine and to formaldehyde was the cause of 80 per cent of the occupational dermatitis in this plant. Hypersensitivity to phenol was also found, but in lesser degree than to formaldehyde and hexa.

The actual cause of dermatitis from exposure to hexa and formaldehyde is the same. They both finally decompose in the presence of warmth and moisture

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Dermatitis from the dust of the molding resins usually occurs at the points of friction with the clothing—the wrist, the belt line, the shoe tops and the collar line. It may, however, occur on the covered parts when the dust penetrates the clothing. The skin around the eyelids may also be affected. The eruption consists, usually, of scattered papules and vesicles on an erythematous base.

It gets well if the worker stays away from work, and returns again if he resumes work. In this factory, the dermatitis was more prevalent in the winter because the workers did not take showers in cold weather after their shift, whereas in the summer they were more likely to do so.

UREA-FORMALDEHYDE RESINS

These are made by mixing urea and formaldehyde in a kettle. No heat is required, the reaction generating its own heat. A syrupy liquid results. This is mixed with bleached sulphite pulp, under heat and pressure, and dried in tray dryers. It is then mixed in a ball mill with pigments, zinc stearate, and a small amount of hexamethylenetetramine, about $\frac{1}{2}$ per cent. It is then screened, and is ready for shipment. Minute amounts (fractions of 1 per cent) of other ingredients are added to different brands.

The manufacture of the urea-formaldehyde resins on a commercial scale is more recent than that of the phenol-formaldehyde resins, hence the two factories studied had more modern machinery and safety appliances than the phenol-formaldehyde resin factories. There were adequate ventilation facilities and good facilities for dust prevention. As a result, among 190 employees over a period of over 2 years, there had occurred only 4 cases of dermatitis. They were all due

to hypersensitivity to formaldehyde. Two chemists in the experimental laboratory of these plants were hypersensitive to formaldehyde and suffered with dermatitis when exposed to it. One of them exposed his forearm in my presence to the mouth of an open bottle of formalin and almost immediately an erythema developed on the exposed skin.

In a molding plant in Ohio employing about 300 workers, where phenol-formaldehyde and urea-formaldehyde resins are used, 26 cases of dermatitis occurred in the first 10 months of 1934, and in a molding plant in Pennsylvania half of the workers developed dermatitis in the hot months of 1935.

The process of molding is practically the same for all the resins. The powder is placed in a "pill machine" and pressed into proper shapes for the molds. The pills are then placed into the molds where they are subjected to heat and pressure which shapes and hardens them. During the molding process gases are given off from the molds and the odor of formaldehyde is strong in the room and irritates the nose and throat and eyes of those unaccustomed to it.

There is an excess of powder in the molds which flows out during the molding process and is only partially "cured." This is called the "flash." When the molds are opened the flash is cleaned off the molds and filed off the molded object.

Patch tests with the resins with which they worked were performed in the winter on 2 active cases of dermatitis, and 10 workers who had recovered from dermatitis, gave only 2 positive reactions, 1 to the phenol-formaldehyde and 1 to the urea-formaldehyde resin. The examination and history of the 10 cases who had dermatitis but were free of it when the patch tests were made, showed that 2 had dermatophytosis and 1 probably had pityriasis rosea. It

is probable that 2 of the 26 cases were not of industrial origin.

The same plant in August, 1935. The dermatitis had occurred 9 months before. The workers who had dermatitis were patched with urea-formaldehyde resin. No reactions after the 7 patches and positive reactions to the resins, and a positive reaction to tetramine.

Two of the 4 reactions had eruption patches were applied of dermatophytosis eczema which dated from the weather was during the period and this may have reactions, because reported in 1934. 15 periods of hot weather, July, and August.

There can be considerable percentage of dermatitis in this plant of origin.

That hexamethylenetetramine is a major causative factor in fact that most of the workers handling urea-formaldehyde resin, which is more than does the urea.

Hexa, which was used in the rubber industry and caused dermatitis almost entirely as an accelerator. It is phenol-formaldehyde resin in order to furnish a hardener of formaldehyde in order to go through the completion.

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is probable that a considerable number of the 26 cases reported in 1934 were not of industrial origin.

The same plant was visited in August, 1935. Twelve cases of dermatitis had occurred since the first visit 9 months before. This time 9 workers who had dermatitis during this period were patched with suspected samples of urea-formaldehyde and phenol-formaldehyde resins, as well as with hexamethylenetetramine. Four showed no reactions after 24 hours to any of the 7 patches applied. Five showed positive reactions to one or more of the resins, and of these 5, 3 showed positive reactions to hexamethylene-tetramine.

Two of the 4 cases who showed no reaction had eruptions at the time the patches were applied. One was a case of dermatophytosis and the other an eczema which dated back to childhood. The weather was cold (less than 70°) during the period the patches were on, and this may have a bearing on the reactions, because, of the 26 cases reported in 1934, 18 had occurred during periods of hot weather in May, June, July, and August.

There can be little doubt that a considerable percentage of the dermatitis in this plant was of occupational origin.

That hexamethylenetetramine is a major causative factor, is shown by the fact that most of the cases occurred in workers handling the phenol-formaldehyde resin, which contains more of it than does the urea-formaldehyde resin.

Hexa, which was extensively used in the rubber industry as an accelerator and caused dermatitis, has now been almost entirely displaced by other accelerators. It is necessary in certain phenol-formaldehyde molding resins in order to furnish the necessary amount of formaldehyde and ammonia required to go through the gamma stage—or to completion.

In the urea-formaldehyde resin, hexa acts as a stabilizer to prevent the resin from hardening before it is molded.

The ventilating and dust prevention conditions in the molding plants were poor. There were no suction hoods over the molding machines. The suction hood over the "pill machine" was out of order. There was only window ventilation in the workrooms, and wash-ups or showers after work were not compulsory. With proper dust allaying facilities, such as forced ventilation, suction hoods, wet sweeping of floors after each shift, compulsory showers, protective clothing and ointments, the incidence of dermatitis in this plant could be decreased.

Dermatitis may occur in users of molded phenol-formaldehyde and urea-formaldehyde resin wares if the hexa is not all combined. Theoretically, it is all combined in the completed resin, but practically (as in some imperfectly cured pieces or pieces in which there was too much hexa in the molding compound to be completely taken up), there may be a sufficient amount remaining to cause dermatitis in hypersensitive people. The same thing may be true of the phenol, if not completely combined.

Dermatitis has been reported as due to contact with finished resin products, such as dermatitis of the ear from telephone receivers, and of the hands from varnishes.

In order to show that a dermatitis may have been caused by contact with a resin, it must be established that:

1. The dermatitis followed contact with the resin
2. That portion of the skin which came in contact with the resin was first affected
3. A powdered portion of the suspected resin placed on the clear skin near the eruption in the form of a patch test, gives a positive reaction if left on 24-96 hours

To determine the actual chemical in the resin which caused the dermatitis,

the nature and chemical composition of the resin must be determined, and patch tests performed on the patient with each of the chemicals composing the resin in order to determine to which of them the patient is hypersensitive.

The patches of the chemicals should be in such dilutions as will not cause reaction on the normal skin if allowed to remain on for 24 hours.

A 2 per cent aqueous solution of phenol can remain on the normal skin in the form of a patch test, for 24 hours without causing a reaction.

A 4 per cent solution of formaldehyde can remain on the normal skin, in the form of a patch test, for 24 hours without causing a reaction.

Powdered hexamethylenetetramine may remain on the normal skin, in the form of a patch test, for 24 hours without causing a reaction.

Powdered coumaron resin may remain on the normal skin, in the form of a patch test, for 24 hours without causing a reaction.

Coumaron resin is used in varnishes, adhesives, rubber, paint, chewing gum, lacquers, paper and fabric sizing, printing inks, and waterproofing.

It is made from the crude coal tar distillate, which comes off between 150° and 200° C.

This distillate is re-distilled to remove impurities such as benzol, toluol, zylol, naphthalene and tar acids—and a sharply fractionated naphtha is obtained. The naphtha is dried in lead-lined or stone-ware receptacles by treatment with H_2SO_4 , in the proportion of 3 to 5 parts per 1,000. After standing a while, the acid is drawn off and fresh H_2SO_4 is added slowly and agitated until 3 to 5 per cent of acid is added. The kettle is kept cold during the reaction with brine-cooled refrigeration.

After the reaction takes place, the mixture is allowed to stand until the tar and sludge settle. They are then

removed and the remaining oil is pumped to a neutralizing tank and treated with caustic soda to destroy the remaining acid. The oil is then washed with water to remove the soluble matter, and it is allowed again to settle and the water is removed.

The oil is now distilled and the solvent naphtha is distilled off and the naphthalene is removed by live steam. After this, there remains a heavy oil boiling at 320°–330° C., which is removed from the resin.

COUMARON RESINS

There are no skin hazards, outside of acid and alkali burns, in these operations, because they are totally enclosed. No skin lesions were found in a large plant and the medical records for a number of years showed no cases of dermatitis.

Dermatitis has occurred from coumaron resin in varnish which was used on heddle frames in a cotton mill. The skin on the forearms of the weavers was struck continuously by the moving heddle frames and some developed dermatitis. Patch tests showed that they were sensitive to chlorinated ceresin and coumaron resin in the varnish. The varnish grades of coumaron resin, especially the darker resins, may contain sulphuric acids resulting from the sulphuric acid treatments, if they are not carefully prepared, and it is these acids that cause dermatitis.

SYNTHETIC WAXES

The principal synthetic waxes manufactured in this country are chloro naphthalenes and the chloro diphenyls. They are both used for practically the same purposes—as electric insulators on condensers, as insulators on electric wires, in paints, varnishes, and lacquers, and as oil in transformers.

The chlorinated naphthalenes are made by passing chlorine through

naphthalene and hydrogen atoms or more of the be replaced, form to per chloro nap of the hydrogen at chlorine, the for. The more chlorine the material.

In making chloro diphenyl, $C_{12}H_{10}$ is first necessary, formed by passing molten lead, at a 800° F., where combine to form chloro diphenyl being s.

Diphenyl is a substance. This cylinders and through, replacing or more of the be replaced, forming di chloro diphenyl, $C_{12}Cl_2$, is a by-product of chloro diphenyls solid, up to the

Nonchloro diphenyl under the trade used as an insulator electric wires are also as a de-luster.

In the manufacture of diphenyls, the w a benzol hazard made, as well as inhalation of the

The workers exposed to the diphenyls, in the operation which is being re-distilled, are affected. In addition of the skin in workers exposed to chloro naphthalenes, fumes of these on the face penetrate the skin like lesions to

naphthalene and replacing of the hydrogen atoms with chlorine. One by one the hydrogen atoms can be replaced, forming mono, di, tri, up to per chloro naphthalene, in which 8 of the hydrogen atoms are replaced with chlorine, the formula being $C_{10}Cl_8$. The more chlorine, the more solid is the material.

In making chlorinated diphenyls, it is first necessary to manufacture diphenyl, $C_{12}H_{10}$. This is manufactured by passing benzol, C_6H_6 , through molten lead, at a temperature of about 300° F., where 2 molecules of benzol combine to form 1 molecule of diphenyl, hydrogen being set free.

Diphenyl is a solid, crystalline-like substance. This is melted in closed cylinders and chlorine is bubbled through, replacing the hydrogen. One by one of the hydrogen atoms can be replaced, forming mono chloro diphenyl, di chloro diphenyl, up to deca chloro diphenyl, $C_{12}Cl_{10}$. Hydrochloric acid is a by-product of this reaction. The mono diphenyls are liquid or semi-solid up to the hexa chloro diphenyl. Mono chloro diphenyl, $C_{12}HCl_9$, sold under the trade name of Atachlor, is used as an insulator for automobile wires and in condensers, and as a de-lusterer of rayon.

In the manufacture of the chloro diphenyls, the workers are exposed to a hazard when the diphenyl is used as well as to a hazard from the fumes of the fumes of diphenyl.

Workers engaged in chlorinating diphenyl, especially that part of the operation where the crude Atachlor is distilled to remove impurities, are afflicted with an acne-like condition of the skin. This also occurs when workers are exposed to the fumes of the chlorinated naphthalenes or Halowax. These compounds cause acne on the face and neck and may also on the clothes and cause acne to develop on the covered

parts, the shoulders, and the belt-line, and even on the penis. The lesions on the skin resemble acne. They begin as small, pale, elevated papules, many having no openings in them. They develop into hard cyst-like elevations under the skin, some of which go on to suppuration, forming boils. Some of the lesions also occur at the mouth of the follicles and resemble the comedones and pustules of acne vulgaris.

In addition to these skin lesions, symptoms of systemic poisoning have occurred among workers inhaling these fumes. Those working with the chloro diphenyls have complained of digestive disturbances, burning of the eyes, impotence and hematuria. The latter symptom developed among a number of men making amino diphenyl, which is used in the manufacture of a rubber antioxidant. Cases of death from yellow atrophy of the liver have been reported among workers exposed to the fumes of the chloro naphthalenes.

Patch tests performed with Halowax and with the chloro diphenyls have yielded negative results. The skin lesions probably result from the mechanical plugging up of the follicles of the skin with the waxes as the fumes solidify on the skin.* The chlorine present in the waxes may have an irritating effect on the plugged follicles and cause suppuration.

PREVENTION

1. The protection of the workers from the irritating chemicals that compose the resins and waxes from the resins and waxes themselves. To do this, the process should be totally enclosed. If this is not possible, hoods with suction exhaust should be so placed over open processes that dust and fumes are pulled away from the worker and out of the room.

*I have recently seen the wife and child of a worker who had developed comedones and pustules from contact with his work clothes which were saturated with halowax and which he was accustomed to wear at home. L. S.

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THE PROBLEM OF POSSIBLE SYSTEMIC EFFECTS FROM
CERTAIN CHLORINATED HYDROCARBONS*CECIL K. DRINKER, MADELEINE FIELD WARREN AND GRANVILLE A.
BENNETT*Department of Physiology, Harvard School of Public Health and Department of Pathology,
Harvard Medical School, Boston, Mass.*

THE use of chlorinated naphthalenes and compounds of allied pharmacological possibilities is extremely wide, and with the steady growth of the use of electricity is certain to expand much farther. For years it has been known that many of these compounds cause a troublesome acne, and there is a large literature upon this phase of the subject. Our investigations have not been concerned with chloracne but with the possibility of systemic effects following ingestion or inhalation of such products. In the spring of 1936, the Halowax Corporation, a division of the Bakelite Corporation, called our attention to three fatal cases of jaundice in workmen using chlorinated naphthalenes and chlorinated diphenyl, and requested that the subject be investigated as

rapidly and thoroughly as possible.† In brief these cases were as follows:

Patient 1. Male, age 21. The previous medical history of this man was in no way significant except for the fact that he had an attack of jaundice about 6 weeks prior to his fatal illness. Late in December, 1936, he became badly constipated and had much abdominal pain and distention. When admitted to the hospital he was slightly jaundiced and was evidently very ill. He was somewhat anemic and his skin, particularly upon the arms, face, chest and back, showed many pustules. He died after a brief period in the hospital, and at autopsy was found to

† The Halowax Company makes many products besides chlorinated naphthalenes, and it has come to our knowledge that all of these products are indiscriminately called "halowaxes" by purchasers and users, and are lumped together as possible causes of acne and even of systemic disease. Since "halowax" is merely a trade designation, care should be taken to describe compounds by their chemical names and thus avoid condemnations which are both troublesome and misleading.

* Received for publication June 30, 1937.

have a cirrhosis of the liver with acute yellow atrophy superimposed upon it. This man had been exposed to low concentrations of vapors arising from a mixture of tetra and pentachloronaphthalenes, together with approximately 10 per cent of a refined chlorinated diphenyl. While both he and others engaged in the same work had chloracne, there were no other disturbances of health in fellow workmen, nor was there any precipitating cause for the acute yellow atrophy such as treatment with arsphenamine or exposure to dangerous concentrations of carbon tetrachloride.

Patient 2. This was a young man who died in February, 1936, after an acute illness characterized by jaundice. He had been exposed to fumes arising from a mixture of penta and hexachloronaphthalenes. There is no record of chloracne. The patient worked with a large number of other people of whom but one (Patient 3), a close friend, had significant illness.

Patient 3. Another young man employed with Patient 2. He became jaundiced in March, 1936, and died after an illness of 2 weeks. A careful autopsy resulted in a diagnosis of acute yellow atrophy of the liver. Here again no history could be obtained as to a precipitating cause, and there was no record of preceding attacks of jaundice.

In addition to these three very recent fatalities, we have learned of four other possible cases, none of them fatal. All of these have had jaundice and the entire group consists of isolated individuals who have been picked out of large groups having the same exposure. In but one instance, Patient 1, is there record of antecedent disturbance of health, and the general health of fellow workers has been good.

Such cases have not been reported in the medical literature and only occasionally can one find reference to systemic effects of any sort. For example, Courtois-Suffit (1934) reports on work done by Touraine and his associates (1934) who examined 60 workers

who had been exposed to trichloronaphthalene. Of these 13 were found to have mild digestive complaints, anorexia, nausea and vertigo, but Courtois-Suffit remarks finally, "Absorption is certainly possible and we have for proof of it some of the digestive and general complaints which have been due to it. But they appear to be of little consequence considering the mildness of the digestive troubles and the absence of respiratory phenomena."

In Touraine's cases the exposure was to a trichloronaphthalene, whereas the American cases of acute yellow atrophy were exposed to compounds of higher chlorination. Our own experiments indicate that trichloronaphthalenes require enormous dosage, far beyond anything encountered in industry, in order to produce liver damage. Tel'ky (1927) reported a number of cases of chloracne in persons exposed to chlorinated naphthalenes with a chlorine content ranging from 14 to 53 per cent. He found that the lower the chlorine content the less the acne. Mittelstädt (1935) examined a number of cases of chloracne due to trichloronaphthalene and reported a number of vague general complaints but nothing in the nature of serious disease. Regarding his animal experimentation, Lehmann (1919) reported that animals fed chlorinated naphthalenes refused to eat after a time and that, whether poisoned by inhalation or by feeding, at death showed "peculiar lesions in the liver. Flinn and Jarvik (1936) gave subcutaneous injections of enormous doses of chlorinated naphthalenes dissolved in paraffin oil to rabbits. The compounds used were as follows:

1. A mixture of tetra and pentachloronaphthalene.
2. A mixture of penta and hexachloronaphthalene.
3. A mixture of penta and hexachloronaphthalene.

In addition, subcutaneous injections (3) were collected and analyzed subcutaneously.

None of the animals receiving the sublimate from 2 died after 2 months were kept and autopsied. The first animals receiving 3 died on the 3rd day and the last died on the 10th day. Those receiving the sublimate were even more ill. Autopsy in these cases showed striking changes in the liver, described, but not as acute yellow atrophy. It is suggestive to consider that these naphthalenes or impurities in them are capable of producing atrophy of the liver.

At the beginning of this paper Flinn and Jarvik (1936) state the fact that there have been cases of acute yellow atrophy in men working with chlorinated naphthalenes but give no reference to them. Their cases are the same as those reported at the beginning of this paper.

One may summarize the literature upon systemic effects of these substances as follows:

1. With the exception of the case of acute yellow atrophy in men working with chlorinated naphthalenes (1936), there are no even suggestions of systemic effects upon human beings.
2. There is evidence that the degree of chlorination

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1. A mixture of tri and tetrachloronaphthalene.
2. A mixture of tetra and pentachloronaphthalene.
3. A mixture of penta and hexachloronaphthalene.

In addition, sublimates from (2) and (3) were collected in oil and injected subcutaneously.

None of the animals receiving (1) or the sublimate from (2) died, and even after 2 months were quite normal when autopsied. The first death in the animals receiving (3) occurred on the 12th day and the last died on the 26th day. Those receiving the sublimate from (3) were even more severely affected. Autopsy in these animals revealed striking changes in the liver, not, as described, entirely characteristic of acute yellow atrophy but sufficiently suggestive to cause the authors to conclude that "certain chlorinated naphthalenes or impurities contained in them are capable of producing yellow atrophy of the liver in the rabbit."

At the beginning of their paper, Flinn and Jarvik (1936) mention the fact that there have been three cases of acute yellow atrophy of the liver in men working with chlorinated naphthalenes but give no details in regard to them. These cases are undoubtedly the same as those described in the beginning of this paper.

One may summarize the meagre literature upon systemic effects from these substances as follows:

1. With the exception of the mention of acute yellow atrophy by Flinn and Jarvik (1936) there are no reports or even suggestions of serious effects upon human beings.

2. There is evidence (Teleky, 1927) that the degree of chlorination is sig-

nificant in relation to the production of acne. In the work of Flinn and Jarvik (1936) the compounds producing serious liver injury were the most highly chlorinated of those tested, though the chlorine contents as given by analysis vary surprisingly little.

3. There are no published figures upon the amounts of various chlorinated naphthalenes in the air which will produce injury of any sort, and while the work of Lehmann (1919) and of Flinn and Jarvik (1936) point to the liver as a possible site of injury this indication rests upon such extreme dosage as to fail to apply directly to human exposure.

EXPERIMENTAL WORK

In appraising the possible toxicity of any substance met in industry it is first necessary to determine the principal route of absorption. In the case of the compounds under consideration there can be no doubt that inhalation is their chief means of entering the body. They are used hot in a great variety of operations and volatilize in varied degree. They are often applied in solution in such volatile solvents as carbon tetrachloride and toluene. The amounts reaching the air under such circumstances are hardly detectable. It will however be shown that carbon tetrachloride adds to the toxicity of the chlorinated naphthalenes and allied compounds, and if there is possibility of inhaling these compounds in other parts of the factory then inhalation of carbon tetrachloride adds a decided hazard. Under such circumstances solvents such as toluene should be used.

Observation in a number of plants causes us to feel that even though

workmen may be extremely dirty and careless, comparatively little of these waxes is eaten. They are tenacious substances, insoluble in water, and if they get on the hands they stick there and are not transferred to the food.

Skin absorption is the third possible means of entering the body. It may occur but at best must be slight when compared to the steady inhalation of finely divided or gaseous material in the air.

They thrive upon a diet very similar to man, and in the case of these chlorinated compounds it is possible that diet may be very significant. Finally, their normal characteristics have been described so well as to make the detection of abnormalities both easy and certain.

Method of exposure. The inhalation experiments were carried out in four large air-tight wooden boxes, each capable of holding ten rat cages, size

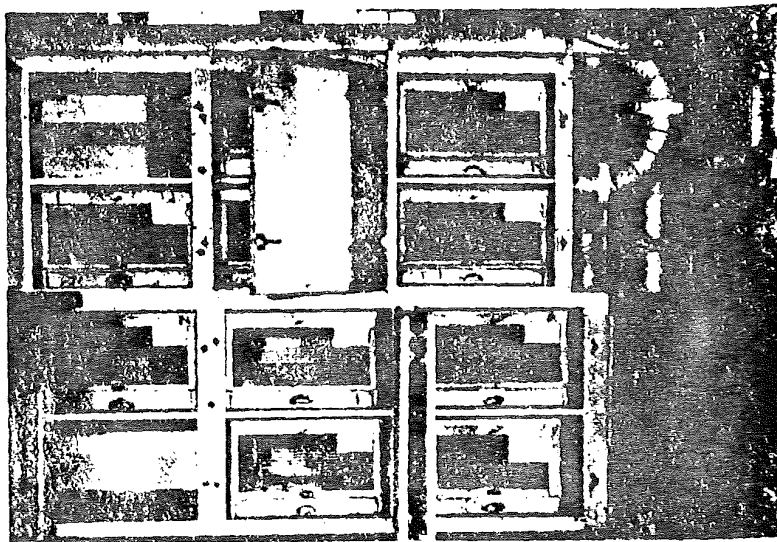


FIG. 1. Front view and inflow end of two boxes with rat cages in place and doors open.

Inhalation experiments are then the most important sources of information, but to them we have added a certain number of observations upon ingestion and subcutaneous injection of various compounds.

Inhalation Experiments

Animals. White rats have been employed throughout. They permit the use of a large number of animals in a relatively small inhalation installa-

tion. They thrive upon a diet very similar to man, and in the case of these chlorinated compounds it is possible that diet may be very significant. Finally, their normal characteristics have been described so well as to make the detection of abnormalities both easy and certain.

At the center of one end of each box the inflow end air was introduced through a pipe 7 inches in diameter (figure 2). Each box was equipped with an individual variable-speed electric blower which blew the air through several feet of 7-inch pipe before enter-

ing the end of the meter was placed 1 ft. from the flow of air in order that the flow could be adjusted, as desired, from a calibration chart. A deflector placed at the end of the pipe into the box was used to insure a uniform stream of air to the cages.

At the opposite end of the outflow end the air was exhausted through a pipe fitted with a damper and a large centrifugal fan.

About 4 inches from the inflow end the 7-inch pipe was fitted with the substance to be introduced into the box. A specially designed holder (figure 4), 7 inches in diameter and 12 inches long, held the test substance. The holders were made with a flange that extended to the top of the flask. This flange was fitted with a screw cap of the same length. The cap was inserted into a large iron pipe and tightly into a hole in the pine on the inside of the flask in turn was a heater made of wire below the side arm. The top of the flask was closed with a side arm, with a reservoir and a gate valve through the side arm motion and a small into each flask. The centrifugal fan was kept in place and the time about 10 to 15 which it passed the

Approximate concentration of chlorinated compounds

ing the end of the box. An orifice meter was placed in the pipe line, and the flow of air in cubic feet per minute could be adjusted and read off directly from a calibration curve. A vane deflector placed at the entrance of the pipe into the box was adjusted to assure a uniform distribution of the stream of air to the two tiers of cages.

At the opposite end of the box—the outflow end—the air from each box was exhausted through a 7-inch pipe fitted with a damper and connected to a large central exhaust fan (figure 3).

About 4 inches from the entrance of the 7-inch pipe into the box, the fumes of the substances listed were introduced into the inflowing air. Specially designed pyrex glass flasks (figure 4), 7 $\frac{3}{4}$ inches long and with a diameter of 1 $\frac{1}{2}$ inches, were used to hold the heated waxes. These flasks were made with a side arm and tube that extended to the bottom. The top of the flask was ground, and into this fitted a short tube 1 $\frac{1}{2}$ inches in length. This short tube was inserted into a large rubber stopper that fitted tightly into a hole cut out of the 7-inch pipe on the under side (figure 2). The flask in turn was placed in an electric heater made to cover it completely below the side arm and ground glass top. Rubber tubing connected the side arm with a compressed air reservoir and a gentle stream of air blown through the melted wax kept it in motion and assured uniform heating. Into each flask was inserted a long-stem centigrade thermometer which was kept in place and could be read at any time above the 7-inch pipe through which it passed (figure 2).

Approximately 30 gm. of pulverized chlorinated naphthalenes or 20 gm. of

chlorinated diphenyl were placed in the bottom of each flask and melted in the electric heater. Fresh samples were used every other day, but it was often found necessary to add 20 gm. of new material even after one run since so much had sublimed. Whatever the case, the collected sublimate was

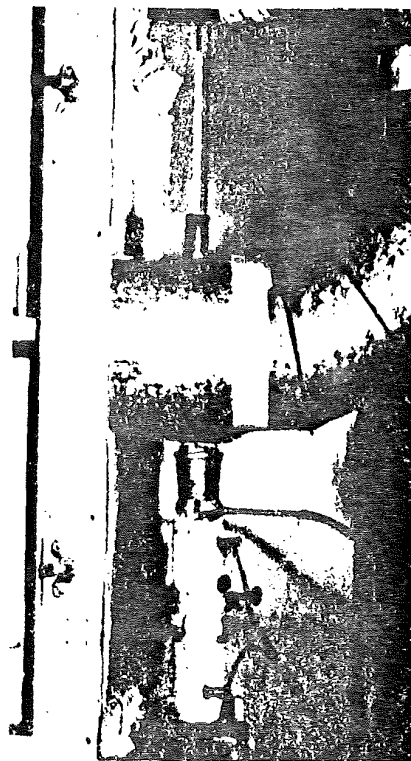


FIG. 2. Electric heater for maintaining chlorinated compounds at a constant temperature in place at inflow end of box.

always removed from the upper part of the flask and a clean top used each day. No sample was ever used for more than two runs.

The flask plus the contents was carefully weighed at the beginning of the run and at the end, and the loss in weight used to calculate the average

amount in a cubic meter of air per minute as determined by a series of

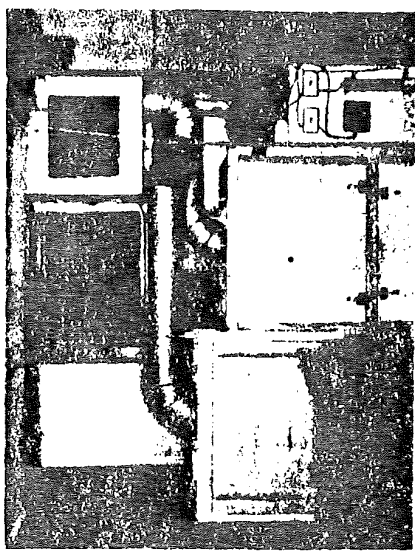


FIG. 3. Outflow end of two boxes showing connections to exhaust system.

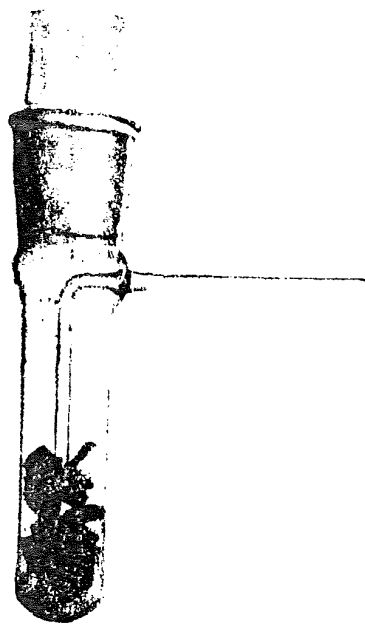


FIG. 4. Pyrex glass flask.

flowmeter readings. The figures obtained were not absolute because of slight variations in the air flowing through the boxes and because of deposition of material on the thermometers and on the inside surfaces of the box, but they checked well with direct determinations through air samples.

Approximately 1 hour was allowed for the wax in the flasks to melt and come to a constant temperature. At that time the box doors were tightly closed, air bubbled through the flasks, and the blowers turned on. This was the beginning of the exposure period. By means of rheostats on the fans and dampers in the outflow pipes, the amount of air flowing through the boxes was adjusted and an attempt was made to keep the four boxes as uniform as possible—usually between 165 and 175 c.f.m.

In the first group of experiments the following substances were tested:

1. A mixture of trichloronaphthalenes plus a trace of tetrachloronaphthalene. Chlorine content 49.9 per cent.
2. A mixture of penta and hexachloronaphthalenes. Chlorine content 62.6 per cent.
3. A mixture of 90 per cent penta and hexachloronaphthalenes plus 10 per cent refined chlorinated diphenyl. Chlorine content 63.0 per cent.
4. Chlorinated diphenyl. Chlorine content 65.0 per cent.

The compounds were selected as representing a certain range in chlorination and also because of their industrial importance. In each instance 80 animals were exposed, 10 rats being placed in each cage. They were fed Purina

vol. 19, no. 5)

Dog Chow rat
eggs, milk and

This first group
began on July
exposure to pure
chloronaphthalene
16th. The exposure
compounds on
18th. On October
five animals were
surre from group
killed after 2
15th, in order to

CONDITIONS MAINTAINED

Trichloronaphthalene
tetrachloronaphthalene

Penta and hexachloronaphthalenes

90% penta and 10% hexachloronaphthalenes plus 10% refined

Chlorinated diphenyl

period would be
the affected group
group 3 were exposed
exposure on October
for examination.
The average daily
16 hours daily
each morning at
ceased, and at
4:00 p.m. the
weighed, etc. The
uniformity of exposure
shifted on a regular

Dog Chow supplemented by lettuce, milk and cod liver oil.

This first group of experiments was begun on July 1, 1936 and the last exposure to number 2 (penta and hexachloronaphthalenes) was on November 16th. The exposure to the other three compounds ceased upon November 18th. On October 15th representative animals were taken out of exposure from groups 1, 2, and 4 and were killed after 2 months on December 15th, in order to see whether this clear

wax concentrations were somewhat higher at the inflow end of the boxes. Preliminary runs showed that once properly adjusted, wax concentrations in the air remained very uniform from day to day, but to insure absolute safety readings of temperature, air-flow, etc. were made every night between 10:00 and 12:00 p.m. as well as on starting and stopping. At different times during the course of the experiment tests for free chloride were made but were uniformly negative and

TABLE 1
CONDITIONS MAINTAINED IN INHALATION EXPERIMENT FROM JULY 1 TO NOVEMBER 16 AND 18

MATERIAL	CHLORINE CONTENT	TEMP.	AV. CONCENTRATION OF AIR IN BOX		TOTAL EXPOSURE	AV. DAILY EXPOSURE
			mg./cu. m.	range	hours	hours
Trichloronaphthalenes plus traces of tetrachloronaphthalene	49.9	150-160	1.31	High 2.60 Low 0.10	1896	16
Penta and hexachloronaphthalenes....	82.6	160-170	1.16	High 2.19 Low 0.51	1864	16
90% penta and hexachloronaphthalenes plus 10% chlorinated diphenyl.	63.0	165-175	1.37	High 3.17 Low 0.64	1896	16
Chlorinated diphenyl.....	65.0	165-175	0.57	High 1.19 Low 0.23	1896	16

period would bring about recovery in the affected livers. Animals from group 3 were similarly removed from exposure on October 4th and killed for examination on December 4th.

The average length of exposure was 16 hours daily for 6 days a week. Each morning at about 9:00 exposure ceased, and between this time and 4:00 p.m. the rats were cleaned, fed, weighed, etc. In order to secure uniformity of exposure the cages were shifted on a regular schedule, since

showed that under the temperatures used no decomposition occurred.

Table 1 shows the temperatures at which the various substances were held, the average concentrations per cubic meter of air, and the average exposures from July 1 to November 16 and 18.

The concentration of chlorinated naphthalenes and chlorinated diphenyl in the air of workrooms.—Table 1 shows that animals have been exposed to varied concentrations of the substances under

test. The concentrations employed may be regarded as fairly representative of industrial exposures. Prior to the initiation of inhalation experiments a number of estimates of chlorinated hydrocarbons in the air of different factories were made and the

in question is passed over heated platinum in an electrically heated quartz tube and the effluent gas scrubbed in a column of glass beads moistened with sodium carbonate containing a trace of sodium sulfite. The beads are then washed down and the chloride deter-

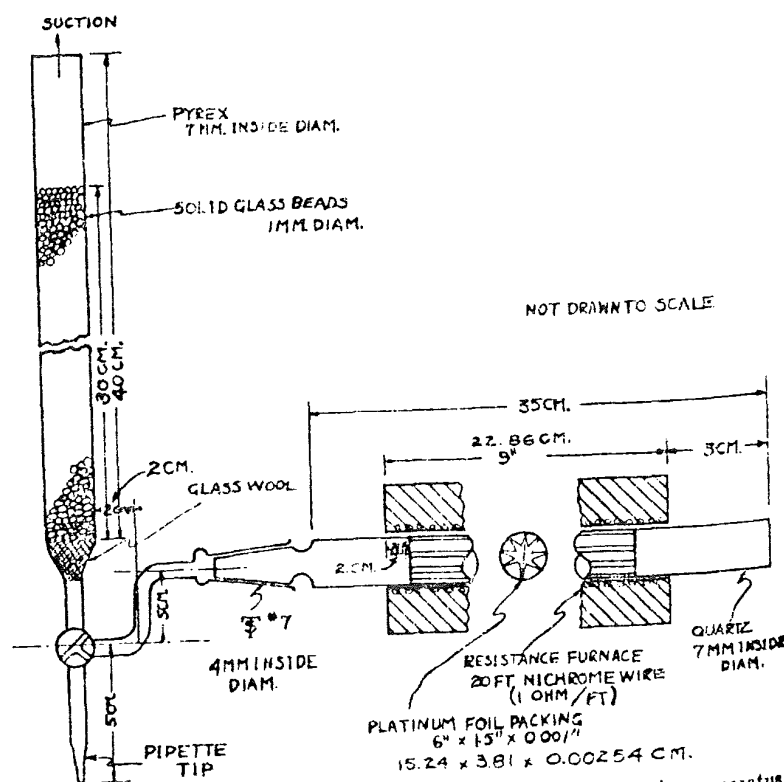


FIG. 5. Dimensioned sketch of combustion tube and absorption apparatus.

concentrations chosen for inhalation experiments depended on these examinations. The technique of analysis and the apparatus employed were the work of Frederick R. Millhiser and William F. Hemperly.

The method and apparatus used for determining concentrations of chlorinated hydrocarbons in air were adapted from well-known procedures. The air

mined nephelometrically as silver chloride.

Tebbens (1937) has recently described a method and apparatus which should be equally satisfactory for this work. In figure 5 we show a dimensioned sketch of the combustion tube and absorption apparatus as used by us in both our laboratory and field problems. The absorption tube is

somewhat easier to use than Tebbens's but the principles of the devices are the same.

In both cases the chlorinated hydrocarbons are absorbed in sodium carbonate solution and subsequent analysis is by nephelometry. In our case concentrations are apt to be very low, less than 1 mgm. per liter, frequently the amount actually formed in the air is so small that it cannot be determined either gravimetrically or by nephelometry. For this reason we have used the nephelometric method which is sensitive to concentrations as low as 0.1 mgm. per liter.

It is doubtful if the conversion of the chlorinated hydrocarbons to hydrochloric acid by subsequent absorption in silver nitrate solution unless the velocity is low. Another problem is that the method does not distinguish between any chlorinated hydrocarbons determined. For this reason it is necessary to distinguish between the gases but determine the results must be computed in terms of total chlorine.

At the present time the formation as to the chlorinated naphthalene is different plants and in some instances the method is repeated several times. The amounts have not been used in our work but must be remembered.

* The combustion apparatus suited to field use by Willson Products. The equipment includes a suction pump with a car-

somewhat easier to wash down than is Tebbens's but the essentials of the two devices are the same.*

In both cases conversion of the chlorinated hydrocarbons to hydrochloric acid and subsequent absorption as sodium carbonate should be complete. In our case concentrations in air are apt to be very low--the objective is less than 1 mgm. per cu. m. Consequently the amount of silver chloride actually formed in the final reaction is so small that it can not be determined either gravimetrically or by titration. For this reason we have been forced to use the nephelometric procedure which is sensitive to concentrations as low as 0.1 mgm. per cu. m. of air.

It is doubtful if the sampling rate should exceed 1 liter per minute as conversion of the chlorinated compound to hydrochloric acid and its subsequent absorption are not efficient unless the velocity of the gas stream is low. Another precaution to be noted is that the method is not selective--any chlorinated substance will be determined. Furthermore it does not distinguish between solid particles and gases but determines them all alike and the results must, of course, be computed in terms of total chlorine.

At the present time we possess information as to the amounts of chlorinated naphthalenes in the air of 30 different plants, and in a number of instances the measurements have been repeated several times. Frequently the amounts have been greater than those used in our experiments, but it must be remembered that the rats

have been exposed for 16 hours to an atmosphere constantly impregnated with the substance under test, whereas human exposure is usually a variable quantity, intense for a short time and then negligible. It is our opinion at the present time that the concentrations of chlorinated hydrocarbons used in our experiments would be dangerous for workers in the case of compounds above trichloronaphthalene in chlorination. Fortunately it is easy to ventilate processes of manufacture which require these substances and to reduce air contamination practically to the vanishing point. Such treatment of the problem at once removes both the possibility of systemic poisoning and the annoyances that arise from cases of acne.

Results of Inhalation Experiments.

1. Animals exposed to a mixture of trichloronaphthalenes plus small amounts of tetrachloronaphthalene. Living animals were apparently entirely normal. Autopsies performed near the end of exposure seemed to show slight swelling of the liver, and microscopic examination occasionally showed swollen and hypergranular liver cells. The changes were, however, never more than slight.

2. Animals exposed to a mixture of penta and hexachloronaphthalenes. No abnormalities were observed in the living animals. Rats were killed and autopsied every 6 weeks. In the first animals sacrificed liver changes were observed. These were swelling of cells, slight granulation and hyalinization. In September and October these conditions were somewhat more advanced, and in November the process became stationary. There were highly

* The combustion and absorption apparatus suited to field sampling is now made by Willson Products, Inc., Reading, Pa. The equipment includes flowmeter and suction pump with a carrying case.

granular cells, hyaline inclusions and mitotic figures, but no more than 2 months previously.

3. Animals exposed to a mixture of 90 per cent penta and hexachloronaphthalenes plus 10 per cent refined chlorinated diphenyl. No abnormalities were seen in the living animals. After 6 weeks the livers showed changes similar to those in the animals exposed to penta and hexachloronaphthalenes. These advanced in grade during August and September and then became stationary.

4. Animals exposed to chlorinated diphenyl. No abnormalities were seen in the living rats. After 6 weeks' exposure there was slight liver damage which advanced during the next 2 months. The changes consisted in slight to moderate swelling of the liver cells, an increased granularity and many mitotic figures. Hyalinization was always present as a result of inhalation of chlorinated diphenyl.

Summary of the first inhalation experiment.—In these experiments careful observation of appearance, body weight, activity, blood, and urine showed no abnormalities of any sort. Yet after 6 weeks' exposure to the compounds with chlorinated diphenyl, trichloronaphthalene caused minor degrees of liver damage, and no changes whatsoever in other organs. There was no acute yellow atrophy or anything suggesting it except that a slight degree of liver damage was always present and was quite clear in the liver sections examined microscopically. This damage had no detectable effect on the health of the animals. They held their weight, ate and behaved normally, being in every respect similar to the many people who have been

exposed to these compounds without illness of any sort.

The functional appraisal of the liver damage caused by certain chlorinated naphthalenes and by chlorinated diphenyl.—There are no tests of liver function useful in such minor degrees of liver damage as were produced in these experiments. Indeed the animals resisted the injury so perfectly as to display no abnormalities except upon histological examination of the liver. The situation was perhaps similar to that met in industry, where, barring acne, the health of workers in these compounds has been good with the exception of the fact that in isolated instances jaundice has occurred which upon at least three occasions has gone on to acute yellow atrophy.

During recent years this disease has been seen following administration of carbon tetrachloride, arsphenamine and cinephen. In the case of carbon tetrachloride it is known that a low calcium diet and alcohol favor the production of liver damage. For the acute yellow atrophy that occasionally complicates use of the other two drugs no cause can be assigned. One cannot produce acute yellow atrophy with arsphenamine, but somehow or other this now and then does happen to patients under antisyphilitic treatment.

It occurred to us that something of the same sort might be involved in this problem. The human cases have been scattered and few. They have been isolated instances out of large groups of healthy employees who have had equal degrees of exposure. It was our idea that perhaps many of these people got liver changes such as existed in our rats, changes not recognizable through

any means other than histological examination. If, upon such changes, there is a disturbance, it is not a very serious one, which any of our rats has over to acute yellow atrophy.

Knowing that the rats had liver changes, slight to cause concern, we decided to test substances known to be destructive. Carbon alcohol were selected on the information that 100 g. of carbon chloride plus 100 g. of alcohol per kilogram of normal white rats is to 0.75 cc. of carbon chloride plus 0.75 cc. of alcohol.

This mixture was added to the food of the rats and with the following table of results.

Rats fed 0.75 cc. of carbon tetrachloride and 0.75 cc. of alcohol.

1. *Trichloronaphthalene and 10 per cent refined chlorinated diphenyl.*
10 1936 10 rats
at 9:30 a.m., Nov. 12
5 died Nov. 12
10:30 p.m.
1 died Nov. 12
1 died Nov. 12
1 died Nov. 12
11:30 p.m.
1 died Nov. 12
11:30 p.m.
1 died Nov. 12
16th (Sat.)
2. *Penta and hexachloronaphthalenes and 10 per cent refined chlorinated diphenyl.*
Fed at 10:00 a.m., Nov. 12

any means other than autopsy examination. If, upon the substratum of such changes, they got an acute liver disturbance, acute catarrhal jaundice, not a very common disease but one which any of us may experience, would this relatively innocuous disease go over to acute yellow atrophy?

Knowing that our inhalation rats had liver changes, but changes too slight to cause recognizable symptoms, we decided to test their resistance to substances known to cause liver destruction. Carbon tetrachloride and alcohol were selected. Having information that 1.0 cc. of carbon tetrachloride plus 1.0 cc. of ethyl alcohol per kilogram would kill 14 per cent of normal white rats, we reduced the dose to 0.75 cc. carbon tetrachloride and 0.75 cc. of ethyl alcohol.

This mixture was given by stomach tube to the following groups of animals and with the results found in the following tabulation.

Rats fed 0.75 cc. per kgm. each of carbon tetrachloride and ethyl alcohol by stomach tube

1. *Trichloronaphthalene plus trace of tetrachloronaphthalene.* Fed at 9:00 a.m., Nov. 10, 1936. 10 rats. No deaths.
2. *Penta and hexachloronaphthalenes.* Fed at 9:30 a.m., Nov. 10, 1936. 10 rats.
 - 3 died Nov. 11th between 4:30 and 10:30 p.m.
 - 1 died Nov. 12th at 3:20 p.m.
 - 1 died Nov. 12th at 4:30 p.m.
 - 1 died Nov. 13th between 9:30 and 11:30 a.m.
 - 1 died Nov. 15th between night of Nov. 14th and 12:30 p.m. Nov. 15th. 2 died Nov. 16th between 10:15 p.m. Nov. 15th and 9:00 a.m. Nov. 16th (stiff).
3. *90 per cent penta and hexachloronaphthalenes and 10 per cent chlorinated diphenyl.* Fed at 10:00 a.m., Nov. 10, 1936. 10 rats.

- 1 died Nov. 10th between 5:00 and 10:30 p.m.
- 5 died Nov. 11th between 4:30 and 10:30 p.m.
- 1 died Nov. 11th between 4:30 p.m. and 9:00 a.m. Nov. 12th.
- 2 died Nov. 12th about 1:00 a.m.
- 1 died Nov. 13th between 5:00 and 10:00 p.m.
4. *Chlorinated diphenyl.* Fed at 10:00 a.m., Nov. 10, 1936. 10 rats.
 - 1 killed Nov. 10th at 10:30 p.m. (almost dead).
 - 2 died Nov. 11th between 4:30 and 10:30 p.m.
 - 1 died Nov. 12th before 8:30 a.m. (stiff).
 - 1 died Nov. 13th between 9:30 and 11:00 a.m.
 - 1 died Nov. 16th between 10:15 p.m. Nov. 15th and 9:00 a.m. Nov. 16th (stiff).
- Controls:* Fed at 9:30 a.m., Nov. 12, 1936. 10 rats. 1 killed Nov. 13th for normal liver.

This tabulation summarizes into the facts that:

1. No normal rats were killed by carbon tetrachloride and ethyl alcohol.
 2. No trichloronaphthalene exposed rats were killed, and this finding agrees with our inability to find lesions of moment in the livers of the animals that inhaled this substance.
 3. The penta and hexachlorinated naphthalene, the mixture of these with 10 per cent chlorinated diphenyl, and finally the latter alone seem to have produced lesions lowering the resistance to an agent capable of producing serious liver disease.
 4. The sole lesion produced by this test of liver function was acute yellow atrophy usually accompanied by jaundice.
- This test of liver function was accomplished with a substance which itself is an organic chloride and curiously enough it is the only substance we were able to find that was effective.

In animals with the minor degree of liver injury which has been described we were unable to produce acute yellow atrophy or any variety of liver effect with arsphenamine, cinchophen, butyl chloride, ethylene chloride and tetrachlorethylene, but carbon tetrachloride was uniformly effective in disclosing the existence of liver damage.

Recovery from liver damage following removal from exposure.—Animals exposed to trichloronaphthalenes plus traces of tetrachloronaphthalene being practically normal on removal from exposure need no consideration. In the case of the penta and hexachlor-

signs of liver injury will be many months in returning to normal.

The effect of high concentrations of trichloronaphthalenes with traces of tetrachloronaphthalene.—The first group of inhalation experiments showed that this material in concentrations averaging 1.31 mgm. per cu. m. of air was relatively innocuous, judged both by direct observation and by the carbon tetrachloride test. This is an interesting fact since such compounds cause acne, though less potently than substances of higher chlorination. In order to explore the matter further one of the inhalation boxes was arranged

TABLE 2
CONDITIONS MAINTAINED DURING INHALATION OF HIGH CONCENTRATIONS OF
TRICHLORONAPHTHALENES PLUS TETRACHLORONAPHTHALENE

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TOTAL EXPOSURE	AV. DAILY EXPOSURE
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in others this did not develop. Fifty rats were used in this experiment and one may conclude that while the trichloronaphthalenes are in no degree as toxic as those of higher chlorination they are not entirely free from effects upon the liver if high enough concentrations are inhaled over long periods.

The effects of high concentrations of penta and hexachloronaphthalenes.—There can be no doubt as to the damage done the liver by these compounds but in the concentrations so far reported no symptoms were caused that could be recognized clinically and nothing approaching acute yellow

the liver after 35 days of freedom from inhalation. It is thus evident that penta and hexachloronaphthalenes are markedly toxic compounds and that recovery from their effects is extremely slow.

The effects of penta and hexachloronaphthalenes, the mixture of these with 10 per cent chlorinated diphenyl, and of chlorinated diphenyl alone, when inhaled in low concentrations through an 8-hour day instead of a 16-hour day as in the first experiment.—Since steady human exposure to any of the compounds tested would invariably be for 8 hours rather than 16-hour periods, a further

TABLE 3
CONDITIONS MAINTAINED DURING INHALATION OF HIGH CONCENTRATIONS OF PENTA AND HEXACHLORONAPHTHALENES

MATERIAL	TEMP.	AV. CON- CENT.		TOTAL EXPOSURE	AV. DAILY EXPOSURE
		mg./cu. m.	range	hours	hours
Penta and hexachloronaphthalenes	137-195	8-85	High 14.0 Low 5.75	608	16

atrophy occurred except through the use of the carbon tetrachloride test. On December 1, 1936, 80 rats were subjected to the conditions summarized in table 3. This experiment was terminated on January 21. All of the animals lost weight and appetite and deaths began after 8 days of exposure. Fifty-five rats died, most of them heavily jaundiced, 8 were killed for microscopic examination, 9 were killed by the carbon tetrachloride test and 8 lived through the period of exposure. The livers of the animals killed for examination showed marked fatty degeneration, central in type with necrosis of liver cells. Surviving animals showed pronounced changes in

inhalation experiment was arranged under the conditions shown in table 4 which are quite comparable to those in table 1 except for the shorter inhalation periods. Eighty rats were used for each of the three compounds tested. None at any time showed the slightest evidence of illness. Microscopic examination beginning after 6 weeks' exposure showed swollen liver cells, excess granulation, hyaline inclusions and occasional mitotic figures. These changes did not progress and were very similar to those in the animals exposed for 16 hours under the conditions set forth in table 1. The carbon tetrachloride test was uniformly fatal to them and one must conclude that con-

centrations of these compounds such as were employed cause a certain degree of liver damage even if inhaled for but 8 hours daily. This injury is resisted successfully by the rats just as was the 16-hour injury but it is none the less there and destroys the ability of the animal to resist the carbon tetrachloride test.

Summary of inhalation experiments. The findings that have been described briefly will be amplified upon the pathological side in a further paper by Dr. G. A. Bennett. What has been given is, however, sufficient to indi-

men never inhale enough of any of these substances to get acute yellow atrophy. They may, however, acquire a substratum of liver damage upon which acute yellow atrophy may develop. Experience in a number of plants has shown how easy it is to reduce concentrations of these compounds practically to the vanishing point, and every effort should be made to attain such conditions.

Gross Feeding Experiments

The various compounds used were ground as finely as possible and mixed with a standard balanced ration for

TABLE 4
CONDITIONS MAINTAINED DURING INHALATION OF LOW CONCENTRATIONS OF THREE COMPOUNDS DURING 8-HOUR INSTEAD OF 16-HOUR PERIODS

MATERIAL	TEMP.	AV. CONCENTRATION OF AIR IN BOX		TOTAL EXPOSURE	AV. DAILY EXPOSURE
		mg./cu. ft.	range	hours	hours
Penta and hexachloronaphthalenes.....	149-193	1.44	High 2.58 Low 0.42	920	8
90% penta and hexachloronaphthalenes plus 10% chlorinated diphenyl.....	150-197	1.66	High 3.30 Low 0.56	912	8
Chlorinated diphenyl.....	153-174	0.93	High 3.23 Low 0.03	920	8

cate that compounds more highly chlorinated than trichloronaphthalene are capable of causing liver injury when inhaled steadily in quite low concentrations. It is an extraordinary thing that even the most searching examination fails to show injury in any other region. It is not easy to grade the toxicity of the different compounds tested, but the chlorinated diphenyl is certainly capable of doing harm in very low concentrations and is probably the most dangerous. Industrial experience combined with these experiments make it appear probable that work-

men never inhale enough of any of these substances to get acute yellow atrophy. They may, however, acquire a substratum of liver damage upon which acute yellow atrophy may develop. Experience in a number of plants has shown how easy it is to reduce concentrations of these compounds practically to the vanishing point, and every effort should be made to attain such conditions.

The experiments were successful in both respects, and indeed one may anticipate that future appraisals of the possible toxicity of chlorinated hydro-

carbons may be made. The feeding and breathing apparatus is necessary for inhalation experiments. In the identification of the compounds by both methods, different compounds seem to arrange themselves in the same way, so far as to be identified by either way they are used.

Trichloronaphthalene. The rats were fed on May 4, 1936, until November 3, 1936, receiving 3 gm. of the compound in the food. The rats were then killed and this was repeated throughout the experiment. The animals were then killed, which died on the loss of weight, wasting illness, death was the respiratory infection to the heart.

Histological examination showed slight changes of great significance.

Tetra chloronaphthalene. Feeding began with a dosage of 0.5 gm. All the animals were either killed or died on 29, 1936.

At autopsy, logically the heart was affected. The lungs, hyperemia, and there were no other changes. There was a tremor.

Penta chloronaphthalene. Feeding began with a dosage of 0.5 gm.

carbons may often be made by simple feeding and do not require the elaborate apparatus and the expense necessary for inhalation experiments. The essential reason for this opinion resides in the identity of the lesions produced by both methods, and the fact that the different compounds so far studied seem to arrange themselves identically so far as toxicity is concerned which ever way they are administered.

Trichloronaphthalene plus traces of tetrachloronaphthalene.—Feeding began on May 4, 1936 and continued until November 2, 1936. In the beginning 3 gm. of this mixture were added to the food for 10 rats each day, and this concentration was maintained throughout the experiment. All the animals were killed except the last which died on Nov. 2. There was no loss of weight and no appearance of wasting illness of any sort. The single death was due to some variety of respiratory infection which had no relation to the material inhaled.

Histological examination showed slight changes in the liver but nothing of great significance.

Tetra and pentachloronaphthalenes.—Feeding began on June 29, 1936. A dosage of 0.5 gm. daily was employed. All the animals sickened gradually and were either killed or died by August 23, 1936.

At autopsy both grossly and histologically the liver was the single organ affected. The liver cells showed swelling, hypergranulation, hyaline inclusions and vacuolation. Here and there necrotic cells were found. There was a tremendous accumulation of fat.

Penta and hexachloronaphthalenes.—Feeding began May 4, 1936, a 3 gm. dosage being used. By June 6, nine

rats had died. The last one was obviously ill and was killed for autopsy examination. All animals lost weight from the beginning and were ill. At autopsy the liver alone was affected, the lesions observed being similar to those that have been described for tetra and pentachloronaphthalenes but worse.

90 per cent penta and hexachloronaphthalenes and 10 per cent chlorinated diphenyl.—Feeding began May 4, 1936 on a 3 gm. dosage. On May 16th feeding was stopped but all the animals went on to death, the last dying on June 8th. An autopsy the liver lesions were extremely severe and of the usual type.

On account of the high toxicity of the 3 gm. dosage, four rats were given a 0.5 gm. dose every other day. Feeding began June 24, 1936 and the last animal was killed Sept. 11th. There were no deaths but all the animals lost weight. At autopsy the liver as usual was the single organ affected, the lesions being characteristic and extensive.

Chlorinated diphenyl. Feeding began May 4, 1936 on a 3 gm. dosage and was discontinued on May 10th. Seven of the 10 rats were dead by May 12th. The three remaining rats gained in weight but were sacrificed for autopsy purposes on July 8th. The liver changes began at once. There were no changes in other organs.

A second group of 10 rats was fed a much smaller dose—0.5 gm. every other day. Feeding began May 20, 1936. The first rat died on May 29th and four more before June 24th. The remaining were sacrificed. Those rats that died showed losses in weight, while those sacrificed had recovered

their initial loss in weight and were gaining. The liver lesions were similar to those found in rats fed penta and hexachloronaphthalenes plus 10 per cent chlorinated diphenyl but not so marked.

Summary of gross feeding experiments.—Of the various materials fed rats in large doses trichloronaphthalene plus traces of tetrachloronaphthalene was quite innocuous. Tetra and pentachloronaphthalene showed definite liver damage. Penta and hexachloronaphthalenes caused a similar grade of injury. The addition of chlorinated diphenyl to penta and hexachloronaphthalenes increased the toxicity. Chlorinated diphenyl alone produced liver lesions but in the dosage used was less effective than when mixed with highly chlorinated naphthalenes. In no case did the compounds used produce acute yellow atrophy but the lesions observed indicate this might be possible if one found a dosage which could act for the proper period of time.

Feeding Precise Doses by Stomach Tube

The compounds employed were suspended in gum acacia. In figuring the dosage the total amount a man of 50 kg. would inhale in an 8-hour day assuming an air concentration of 20 mgm. per cu. m. was first calculated and reduced to milligrams per kilogram. The rats and rabbits received this dose each day. The compounds used were those employed in the gross feeding experiments and the results were essentially similar though the lesions were less severe.

Subcutaneous Injections

The same gum acacia suspensions were injected subcutaneously into rats

and rabbits, the dosage being calculated on the basis of 4 mgm. per cu. m. of air. Again similar results were obtained. In all such experiments there must of necessity be differences in the degree of effect but invariably the liver was the sole organ affected and the lesions were those already described many times.

DISCUSSION

These experiments leave no doubt as to the possibility of systemic effects from the chlorinated naphthalenes and chlorinated diphenyl. As in the case of the effects upon the skin, the degree of chlorination seems to determine the systemic toxicity, and it is a striking thing that when trichloronaphthalene is reached systemic effects are never marked and are produced with the greatest difficulty. It is most remarkable, too, that all the compounds tested attack the liver and the liver alone. During the past few months we have determined the organically combined chloride in the livers of animals very severely poisoned by penta and hexachloronaphthalenes but have found no increase over normal figures, though the livers, as determined histologically, were very severely affected. At the present time we are conducting inhalation experiments on a chlorinated diphenyl containing 55 per cent of chlorine instead of 64 per cent as in the case of the experiments reported in this paper and on a compound with a chlorine content between tri and tetrachloronaphthalene. We are also determining the degree to which the diet may increase or decrease toxicity, this being suggested by similar work upon carbon tetrachloride.

In the basis of these experiments

and on many fields with different compounds in rooms, it appears certainly easily attained that the air breathed is more than 0.5 mgm. per of these compounds of naphthalene. In these compound concentrations are permissible. We many examinations in plants that such concentrations have been greatly exceeded for 20 years, and we are not fact that our results have been inexorably against human exposure is not. Time and careful study change these opinions, but today we are convinced safe. Impregnation and arrangements with hydrocarbons are a safeguard. Compounds lead tetrachloride and

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and on many field determinations of different compounds in the air of work-rooms, it appears safe and it is certainly easily attained, to ventilate so that the air breathed does not contain more than 0.5 mgm. per cu. m. of any of these compounds above trichloronaphthalene. In the case of the latter compound concentrations of 10.0 mgm. are permissible. We know from many examinations in many different plants that such concentrations have been greatly exceeded during the past 20 years, and we are conscious of the fact that our rat exposures have been inexorably constant whereas human exposure is never so ordered. Time and careful observation may change these opinions as to standards, but today we are convinced they are safe. Impregnating tanks and other arrangements utilizing the chlorinated hydrocarbons are easy to hood and to safeguard. Compared with benzene, lead tetrachl and many other com-

pounds, these substances are very little toxic and operations employing them can easily be safeguarded. It may be argued that where possible trichloronaphthalene should be used, but this compound will cause acne and if employed very carelessly might do more. Furthermore, higher chlorination is often essential for highly practical reasons. The solution consists in thoroughly adequate ventilation plus good housekeeping around all wax containers.

A final word of caution bears upon the use of carbon tetrachloride as a wax solvent. Obviously this compound adds readily to the toxicity of the highly chlorinated carbon tetrachloride is not so good. It should be excellent, but in our opinion it would be better to dispense with carbon tetrachloride and depend on other solvents, especially upon those containing no chlorine.

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**DEPOSITION
EXHIBIT**

#4 5/27/87
L.R.

CYNTHIA M. WEICHMANN
Carol L. Davis
Reporting Service
Houston, Texas

Emme H
Exhibit No. 19

Further studies - hepatic morphology - only
effs liver.
detail on progression of liver histology
from chlorinated biphenyls.

**THE JOURNAL OF
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**MORPHOLOGICAL CHANGES IN THE LIVERS OF RATS
RESULTING FROM EXPOSURE TO CERTAIN
CHLORINATED HYDROCARBONS***

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CHLORINATED hydrocarbons, particularly chlorinated naphthalenes and chlorinated diphenyl, have been used extensively in certain industries. Their use in the manufacture and preparation of many types of electrical equipment is constantly increasing. Although it is known that some of these compounds cause acne, only recently has the possibility of more serious systemic effects been recognized.

During the spring of 1936 we were informed of the occurrence of three fatal cases of jaundice in workmen using chlorinated naphthalenes and chlorinated diphenyl. At the request of the manufacturers of these compounds, we undertook an investigation.

* Received for publication September 8, 1937.

This is the second of three papers read at a symposium on chlorinated hydrocarbons given at the Harvard School of Public Health, Boston, June 30, 1937. The third paper will appear shortly.

to determine what systemic effects, if any, would result from the administration of a number of these compounds to experimental animals.

The known findings in the three fatal cases of jaundice, together with a review of the pertinent literature have already been reported (1).

The present paper describes the pathological changes observed in rats that had been exposed to various chlorinated naphthalene compounds and to chlorinated diphenyl.

MATERIALS AND METHODS

A detailed description of the apparatus and technic employed by the authors in this investigation has been published (1). White rats, maintained on a diet of Purina Dog Chow, supplemented by lettuce, eggs, milk, and cod liver oil, were used throughout the experiments.

The chlorinated hydrocarbons tested were the following:

Compound A. A mixture of tri- and tetrachloronaphthalenes.*

Chlorine content 49.4 per cent.

Compound B. A mixture of tetra- and pentachloronaphthalenes. Chlorine content 58.4 per cent.

Compound C. A mixture of tetra- and pentachloronaphthalenes plus chlorinated diphenyl. Chlorine content 43.5 per cent.†

Compound D. A mixture of penta- and hexachloronaphthalenes. Chlorine content 62.6 per cent.

Compound E. A mixture of penta- and hexachloronaphthalenes. Chlorine content 62.6 per cent.

Compound F. A mixture of 90 per cent penta- and hexachloronaphthalenes, plus 10 per cent chlorinated diphenyl. Chlorine content 63 per cent.

Compound G. Chlorinated diphenyl. Chlorine content 65.0 per cent.

These preparations were selected because of their relative importance in industry. It should be noted that they also represent a wide range of chlorination.

All of the above materials were administered orally in varying doses. Compounds A, D, F, and G were selected for the inhalation experiments.

* This compound contained only small amounts of tetrachloronaphthalene. In this paper it will therefore be referred to as trichloronaphthalene.

† This compound consisted of a mixture of compounds B and G but in addition contained two plasticizers which have been considered to be inert. Without these materials the chlorine content of this compound would be between that of compounds B and D.

Inhalation Experiments

We were most interested in the results of the inhalation experiments because they more nearly simulate the type of exposure to which the workmen are subjected (1). The inhalation experiments were carried out in air-tight wooden compartments through which air containing the volatilized compound being tested, was driven by electrical blowers. With this apparatus we were able to test simultaneously the effect of four compounds. Eighty rats were exposed to each compound. The methods for determining the daily air concentration (mgms. per cu. m.) of the chlorinated hydrocarbon being used, as well as the rate of airflow have been described (1).

Compound A (trichloronaphthalene) was administered by inhalation to two groups of animals. In the first of these experiments, the rats were exposed to low air concentrations (average 131 mgms. per cu. m.) 16 hours daily for 134 days. In the second experiment the concentration was increased to an average of 10.97 mgms. per cu. m. This 16 hour daily exposure was continued for 102 days.

Compound D (hexa- and pentachloronaphthalenes) was administered to three groups of rats. In the first experiment the exposure consisted of 16 hours daily for 134 days, with an average air concentration of 116 mgms. per cu. m. In a second experiment the average air concentration was 144 mgms. per cu. m. This was maintained 8 hours daily for 143 days. In the final experiment, the average air concentration was increased to 8.88 mgms. per cu. m. and exposure, 16 hours daily, was continued over a period of 52 days.

Compound F (hexa- and pentachloronaphthalenes 90 per cent plus chlorinated diphenyl 10 per cent) was administered in low concentrations to two groups of rats. In the first experiment an average air concentration of 1.37 mgms. per cu. m. was maintained 16 hours daily for 134 days. In the second experiment, an average air concentration of 1.66 mgms. per cu. m. was inhaled 8 hours daily for 143 days.

Compound G (chlorinated diphenyl) was administered to two groups of animals in low concentrations. An average concentration of 0.57 mgms. per cu. m. was employed 16 hours daily for 134 days in the first experiment. In the second experiment (employing an average air concentration of 0.93 mgms. per cu. m.) the animals were exposed 8 hours daily for 143 days.

Feeding experiments

All compounds tested were employed in the feeding experiments. A weighed amount of finely ground material was mixed with rat food. This mixture was placed daily in one food container to supply the rats in a given cage. Thus each animal had an equal opportunity to ingest the compound supplied. As the number of rats per cage was reduced, the dosage supplied was proportionately decreased. Most compounds were supplied in large and small amounts.

Supplementary experiments were conducted on a smaller number of rats by feeding several of these compounds in small known amounts by stomach tube or by injecting them subcutaneously as suspensions in gum arabs.

Carbon tetrachloride and alcohol administration to experimental animals

Seemingly the incidence of acute yellow atrophy in workers exposed to chlorinated naphthalenes and chlorinated diphenyl is very low. This single fact is of some importance in that it suggests that certain individuals may be more susceptible to the compounds or that in these instances the liver damage may have been intensified by some other agent. With this in mind, groups of animals that had been exposed for varying periods of time to certain chlorinated naphthalene compounds and chlorinated diphenyl were subsequently given a sublethal dose* of carbon tetrachloride and ethyl alcohol by stomach tube (0.75 cc. of each per kgm.). This dosage did not result in a single death among the 16 control animals and the degree of liver damage produced was quite constant and never very great. Administration of carbon tetrachloride and alcohol to rats exposed to the more highly chlorinated naphthalene compounds and chlorinated diphenyl caused extensive liver damage and proved widely fatal. Therefore, the use of carbon tetrachloride and alcohol has been employed regularly on representative groups of rats from each inhalation experiment.

Examination of tissues

Animals were sacrificed for pathological examination after varying periods of exposure. Complete autopsies were performed. Liver weights were recorded. In representative animals

* One cubic centimeter of carbon tetrachloride plus 1.0 cc. ethyl alcohol per kilogram when administered orally to normal white rats causes a 14 per cent mortality (2).

from each experiment, all organs excepting the central nervous system were examined microscopically. The livers of all animals were studied microscopically. Tissues were fixed in Zenker's fluid, 10 per cent formaldehyde solution, and absolute alcohol. The routine paraffin sections were stained with eosin methylene blue. Other stains employed were hematoxylin and eosin, phosphotungstic acid hematoxylin (Mallory), Foot's modification of Bielschowsky's stain (reticulum), Best's carmine stain, and iron reaction with ferrocyanide of potassium (Mallory). In addition, frozen sections from certain liver specimens were stained with scarlet red, Lugol's solution, and methyl violet.

RESULTS

Compound A. Trichloronaphthalene

(a) Exposure by inhalation to low concentrations (average 1.51 mgms. per cu. m.) 16 hours daily. The 80 rats

subjected to these conditions showed no ill effects. Autopsies were performed on groups of 3 to 8 animals after 37, 72, 105 and 134 days' exposure. There were no significant abnormalities in liver weights.* Macroscopically the majority of the livers appeared normal although an occasional one was paler than normal. Rarely slight mottling was observed. Microscopically, the liver cells often appeared slightly enlarged, more granular than normal, and occasionally they were vacuolated. These slight abnormalities and the presence of a rare mitotic figure suggested that very slight injury to the liver cells had occurred. There was no demonstrable increase in the above changes after the first exposure period (37 days). Occasional livers of rats exposed for the total period showed

* Calculations pertaining to the weights of livers in these and subsequent animals are based on data in H. H. Donaldson's book "The Rat," 2nd ed. The Wistar Institute, Philadelphia, 1924 (p. 211).

PLATE I

FIGS. 1 AND 2. Camera lucida drawings showing the portal areas of the livers of two rats that were fed large doses of a mixture of tetra- and pentachloronaphthalene and chlorinated diphenyl. The changes illustrated in figure 1 occurred within 48 days after exposure was begun. One should note the marked accumulations of hyaline globules in the cell cytoplasm, and the marked swelling of liver cells. Increased numbers of mitotic figures were also present. Similar changes are apparent in figure 2 which was made from the liver of a rat exposed 125 days. Degeneration in the central portions of the liver lobules is also present.

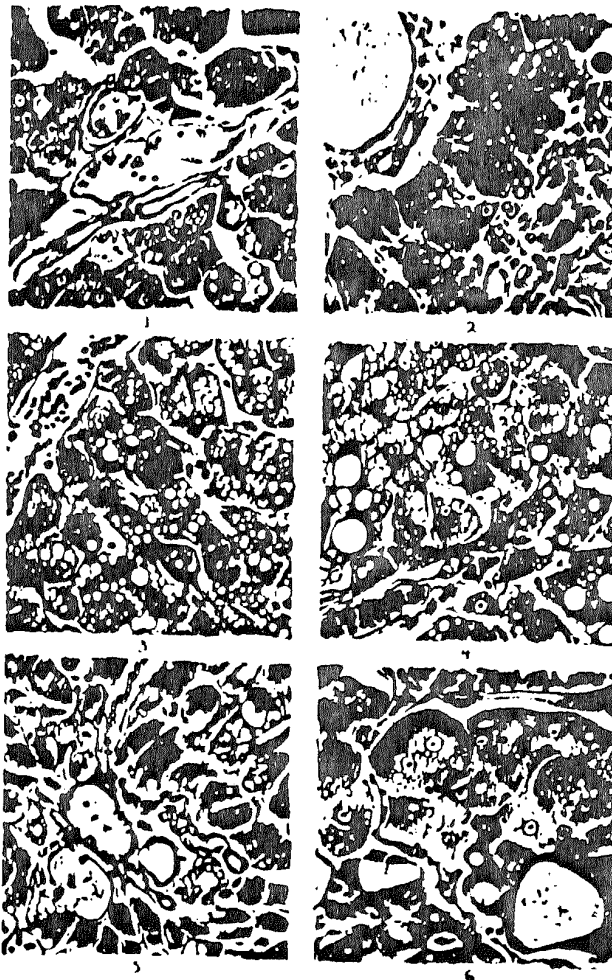
FIGS. 3 AND 4. These drawings illustrate the marked swelling and fatty vacuolation of liver cells observed in all rats that were fed small doses of tetra- and pentachloronaphthalene. The changes shown in figure 3 were present after 26 days' exposure, those in figure 4 after 48 days' exposure. Note the mitotic figures in figure 3. The compound responsible for the changes contained as chlorinated diphenyl. Otherwise

it was similar to the compound responsible for the lesions illustrated in figures 1 and 2.

FIGS. 5 AND 6. Camera lucida drawings of the portal areas of the livers of two rats fed moderate sized daily doses of a mixture of penta- and hexachloronaphthalene for 14 and 29 days respectively. In the first of these figures one notes swelling of liver cells with narrowing and distortion of the sinusoids. The liver cells show an increased granularity of the cytoplasm with a massing of basophilic granules near the nuclei. There is also an excess of fat in small and medium sized vacuoles. This was more prominent in the central portions of the liver lobules. An increased spacing between liver cells and between liver cells and sinusoidal endothelium is present. After 29 days' exposure (fig. 6) the above changes are greatly increased. Numerous large intercellular spaces containing serous precipitate, strands of fibrin and leucocytes had developed between liver cells. These spaces were bounded by distorted liver cells. See also plate III, fig. 1.

Camera lucida drawings $\times 395$.

PLATE I



no definite microscopic change (See fig. 3, plate II). All other organs were normal.*

Further evidence that the liver tissue injury had been very slight was obtained from the carbon tetrachloride and alcohol test. Of 10 rats exposed for 141 days in the above manner and then fed 0.75 cc. per kgm. each of carbon tetrachloride and ethyl alcohol by stomach tube, none died. At autopsy and on microscopic examination there was little or no evidence of liver injury greater than that produced by the same dose of carbon tetrachloride and alcohol in normal control rats. (See plate IV, figs. 1 and 2, and plate V, fig. 1.)

The livers of 8 animals exposed to trichloronaphthalenes for 105 days and autopsied after a 2 month recovery period showed no definite pathological change. This is further evidence that this compound is only slightly toxic. These findings represent a marked

* In none of the experiments to follow were significant changes found in any organ other than the liver.

PLATE II

FIGS. 1, 2, AND 3. Liver changes resulting from the administration of trichloronaphthalene can be illustrated in these camera lucida drawings. In figures 1 and 2 are shown the most marked changes observed after prolonged (100 and 136 days respectively) feeding in daily doses of 4 gm. per 10 rats. The change observed consisted of slight to moderate swelling of liver cells, increased granularity of the cytoplasm and finally, after prolonged exposure, fatty vacuolization of the majority of cells and complete fatty degeneration of occasional cells. Figure 3 is a drawing from a section of the liver of a rat exposed by inhalation to low concentrations of trichloronaphthalenes for 106 days. No striking or constant changes were observed in rats so treated. Occasional cells are slightly swollen and show increased granularity of the cytoplasm. A rare mitotic figure was observed in occasional sections.

FIGS. 4, 5, AND 6. Microscopic changes

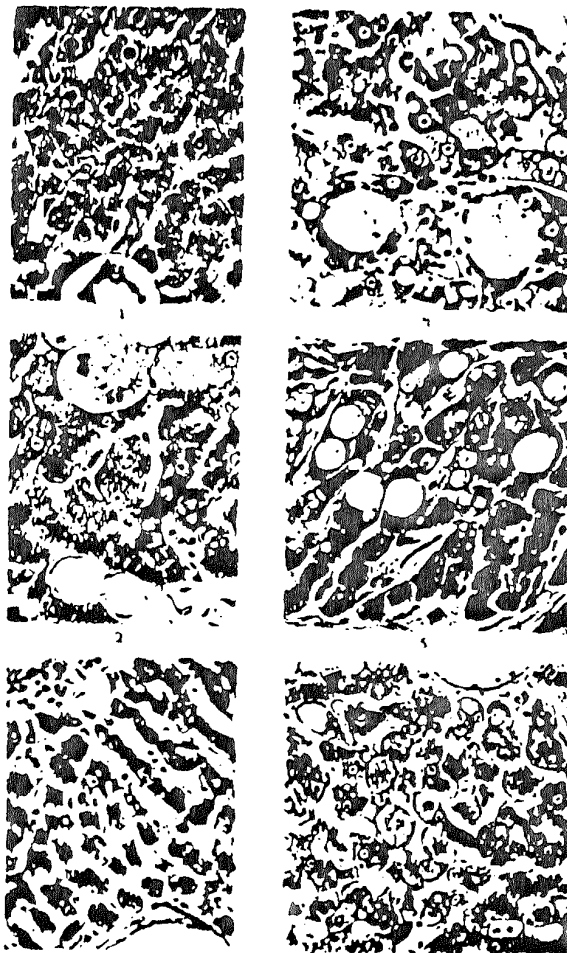
contrast to the persistent hepatic lesions resulting from exposure to the compounds of higher chlorination.

(b) *Exposure by inhalation to high concentration (10.9% mgms. per cu. m.) 16 hours daily.* In this experiment, the concentration of trichloronaphthalenes was approximately eight times that previously employed. Exposure was continued for 102 days. The 50 animals subjected to this concentration appeared normal. Groups of 3 to 17 rats were sacrificed after exposure periods of 31, 48, 59, 72 and 102 days. Most of the livers were pale yellow in color and slightly mottled. There was, however, no significant variation in liver weight. Microscopic examination revealed swollen liver cells with slightly increased granularity and vacuolization of the cytoplasm. Occasional degenerating and regenerating cells were observed. These changes, which were slightly more marked in the central portion of the liver lobules, were present after the first month of exposure. They

observed following the administration of a mixture of pent- and hexachloronaphthalenes are illustrated in these three drawings. Figure 4 shows the changes observed after feeding this preparation in 4 gm. daily doses per 10 rats for a period of 40 days. The liver cells are markedly swollen and vacuolated. There is also necrosis and degeneration of scattered cells. Large intercellular spaces are observed. (See plate VI, figs. 1 and 2.) Less marked but similar changes are observed in the livers of rats fed smaller doses of this preparation for 53 (fig. 5) and 76 days (exposure 1). Figure 6 illustrates the liver cell changes that occurred after inhalation of low concentrations of pent- and hexachloronaphthalenes for 75 days. Note the swelling of the liver cells, the increased vacuolization and presence of large numbers of hyaline globules within cells. Mitotic figures were frequently observed.

Camera lucida drawings $\times 495$.

PLATE II



were somewhat increased with larger exposure. After 102 days the liver cells were more markedly swollen and contained large and small fat vacuoles. Small hyaline droplets in the cell cytoplasm were occasionally observed. Mitotic figures, although never numerous, were increased in number. No structural changes had occurred in the liver lobules.

Carbon tetrachloride and ethyl alcohol were administered by stomach tube to 9 rats kept under these conditions. At autopsy their livers were slightly to moderately enlarged. They were yellow in color and the majority of them were mottled. The microscopic findings were variable. In one animal the observed necrosis was no greater than that seen after the administration of a similar dose of carbon tetrachloride and alcohol to normal control rats. The livers of 4 animals showed increased fatty degeneration in the central portions of the lobules with small necrotic foci. In the 4 remaining animals extensive central necrosis had occurred. In the

most marked of these (fig. 2, plate V), the central one-half or two-thirds of each liver lobule was necrotic and in occasional areas two or more adjacent lobules had undergone degeneration. Hemorrhage and extensive leucocytic infiltration had occurred in these necrotic areas. The less damaged liver cells at the periphery of the lobules showed large numbers of mitotic figures, indicating accelerated regenerative activity.

(c) *Exposure by feeding.* Nine of the 10 rats fed trichlorophthalenes in doses of 3 gm. per day per 10 rats were sacrificed at varying periods of time from 9 to 136 days. The tenth rat died of a respiratory infection after 182 days' exposure. There was no significant abnormality in the weights of the livers of these animals. After 2 months' exposure, microscopic examination of the livers showed slight swelling of liver cells. This was accompanied by increased vacuolization of the cytoplasm due to the accumulation of abnormally large

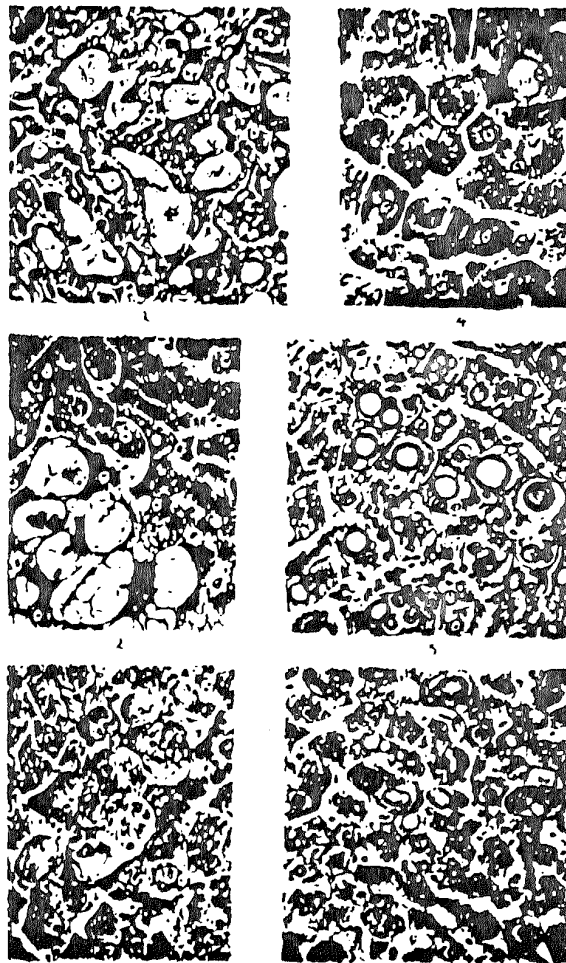
PLATE III

FIGS. 1, 2 AND 3. Liver changes resulting from the administration, by feeding and inhalation, of a mixture of pentachlorophthalene (90%) and hexachlorophthalene (10%) and chlorinated diphenyl (10%) are illustrated in these drawings. Figure 1 is a drawing made from a section stained with P.T.A.H. In addition to other changes it demonstrates the presence of large and small intercellular spaces, interpreted as dilated bile capillaries. These spaces contained serous precipitate, fibrin, and occasional leucocytes. Note the sharply outlined boundaries of the spaces and the manner in which these spaces ramify between cells. The rat was exposed by the feeding of large daily doses for 12 days. Similar but less marked changes resulted from the feeding of small doses over a period of 28 days (see fig. 2). After 75 days' exposure by inhalation to low concentrations of this preparation, marked liver changes, consisting of cellular swelling, vacuolization,

and hyalinization were apparent (fig. 3). FIGS. 4, 5, AND 6. The microscopic changes resulting from exposure to chlorinated diphenyl are illustrated in these figures. Figure 4 was made from the liver of a rat fed large daily doses for only 6 days. Marked swelling of cells and rapid regenerative activity are apparent. Figure 5 illustrates the changes that were uniformly produced by the feeding of small doses of this preparation. Note the extremely numerous hyaline inclusions in liver cells. This rat was exposed for 29 days. Very similar changes were produced by the administration of this compound by inhalation methods (fig. 6). The rat from which this drawing was made had been exposed to low concentrations for a period of 107 days.

FIG. 1. Camera lucida drawing $\times 95$.
FIGS. 2 to 6. Camera lucida drawings $\times 195$.

PLATE III



amounts of fat. These changes increased slightly with longer exposure (See plate II, figs. 1 and 2). There were no architectural changes in the liver.

Because the effects resulting from large doses of trichlorophthalenes were slight, this compound was not fed in small amounts.

Compound B. Tetra- and pentachlorophthalenes

Exposure by feeding. This preparation was employed only in feeding experiments. The daily food ration for 10 rats contained 0.5 gm. of this compound. All animals fell ill and either died or were sacrificed by the sixty-third day.

The livers were pale yellow in color, friable, and occasionally showed mottling. There were no significant variations in weights. On microscopic examination the same changes were observed in all rats. These consisted of moderate to marked swelling and rounding of the liver cells. The sinusoids were markedly narrowed. The majority of the liver cells contained large numbers of small fat vacuoles. This finding was most marked and first observed in the central portions of the lobules. In the least altered cells there was prominent massing of basophilic granules near the nuclei. Occasional necrotic cells were observed. Mitotic figures

were present in increased numbers, indicating accelerated regenerative activity. The microscopic changes resulting from the feeding of tetra- and pentachlorophthalenes are illustrated in plate I, figures 3 and 4. They are those of diffuse fatty infiltration and fatty degeneration.

Compound C. Tetra- and Pentachlorophthalenes plus Chlorinated Diphenyl

Exposure by feeding. Feeding (3 gm. doses daily per 10 rats) was continued with but one 1 day interruption for 130 days. Ten rats were so treated. Seven rats were sacrificed at varying periods of 17 to 124 days. Three rats died after exposure periods of 122 to 130 days. In all animals the livers were enlarged (33 to 90 per cent). The average weight increase was 71 per cent. They were also frangible, pale yellow in color, and somewhat mottled. On microscopic examination, consistent changes were observed. Practically every liver cell was swollen and rounded. Their cytoplasm contained large numbers of hyaline bodies. These were circular or oval in shape and varied in size from about half the size of a red blood corpuscle to twice the size of the nucleus of a liver cell (See figs. 1 and 2, plate I). In numerous instances, many small hyaline bodies had fused, forming large circular masses as large or larger than a normal

PLATE IV

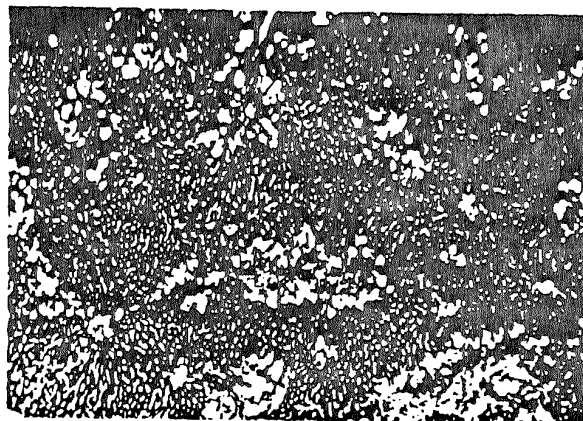


Fig. 1

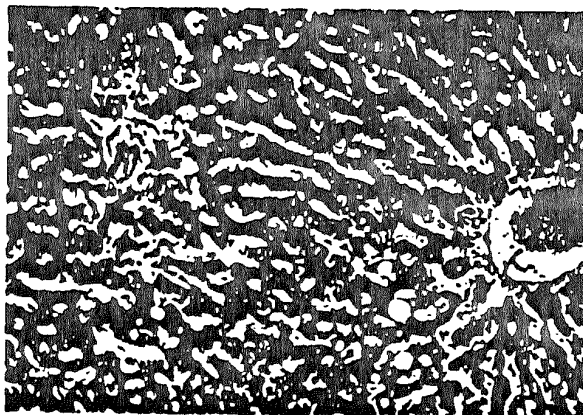


Fig. 2

PLATE IV

Fig. 1. Photomicrograph $\times 15$ illustrating the extent of the liver damage produced in 18 hours by 0.75 cc. per kgm. each of carbon tetrachloride and ethyl alcohol when administered by stomach tube to a normal control rat. The degenerative changes were limited to the central portion of the lobules.

Fig. 2. Photomicrograph $\times 250$ of a

section of liver tissue from a rat exposed by inhalation in the dark to trichlorophthalene in low concentration for 150 days. Following this exposure 0.75 cc. per kgm. each of carbon tetrachloride and ethyl alcohol was administered by stomach tube. The animal survived and was sacrificed 17 days later. No significant liver changes are demonstrable.

liver cell. These bodies stained brilliantly with eosin dye. They were often binucleated and occasionally contained small clear fat vacuoles in their central portions. Mitotic figures in liver cells were sufficiently numerous to indicate an increased rate of regeneration (fig. 1, plate I). In the livers of 2 rats exposed for 124 and 125 days respectively, there were, in addition to the above changes, large areas of complete liver cell degeneration (fig. 2, plate I). For the most part this was limited to the central half or third of the liver lobules. Occasionally, however, there was complete degeneration of all liver cells except for a narrow zone of cells around the portal areas. In such livers there was a heavy polymorphonuclear and mononuclear inflammatory cell infiltration in the necrotic areas where liver cells were being removed.

The most conspicuous feature of the microscopic changes in the livers of these rats was the presence of large numbers of circular hyaline droplets in the cytoplasm of the liver cells. This material did not stain in a manner characteristic of amyloid and its staining properties were not like those of the hyaline observed in alcoholic cirrhosis. In frozen sections stained with Scharlach R, the staining reac-

tion of these bodies was inconstant. The larger vacuolated hyaline bodies contained small droplets of fat while the majority of the smaller droplets did not stain red. Although similar hyaline droplets were observed in livers of rats exposed to various chlorinated naphthalenes, this type of degeneration occurred much earlier and to a much more marked degree in those rats that were exposed to preparations containing chlorinated diphenyl (figs. 1 and 2, plate I) or to chlorinated diphenyl alone (figs. 4, 5, and 6, plate III).

Compound D: Pentachloronaphthalenes

Lat. Exposure by inhalation to low concentrations (average 1.0 mgms. per cu. m.) 16 hours daily. Eighty rats living under these conditions appeared normal throughout the exposure period (131 days). Representative animals were sacrificed in groups of 3 to 15 animals after varying exposure periods as described in the experiments employing trichloronaphthalenes. There were no significant alterations in liver weights. Macroscopically the majority of the livers were light yellow in color and slightly mottled. After the initial exposure period of 37 days there was evidence of slight injury to

PLATE I

Fig. 1. A photomicrograph $\times 120$ showing small areas of necrosis in central areas of the liver lobules. This rat had been exposed by inhalation to low concentrations of trichloronaphthalenes for 131 days following which carbon tetrachloride and ethyl alcohol were administered by stomach tube in doses of 0.75 cc. each per kgm. The rat was sacrificed 5 days later. The extent of necrosis is similar to that resulting from carbon tetrachloride and alcohol alone.

Fig. 2. In this photomicrograph $\times 15$ are shown the most marked liver changes

that were observed following the administration of carbon tetrachloride and alcohol (0.75 cc. of each per kgm.) to rats which had been exposed by inhalation methods to high concentrations of trichloronaphthalenes. This rat had been exposed to trichloronaphthalenes for 4 months. Very extensive necrosis was present in the central areas of each liver lobule. In occasional areas the major portion of several adjacent lobules were necrotic. This rat was autopsied 10 hours after the administration of carbon tetrachloride.

PLATE V

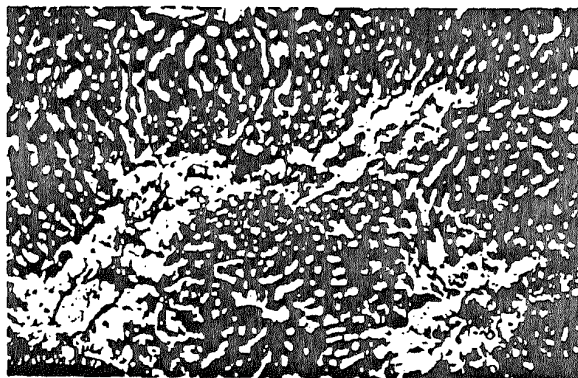


Fig. 1

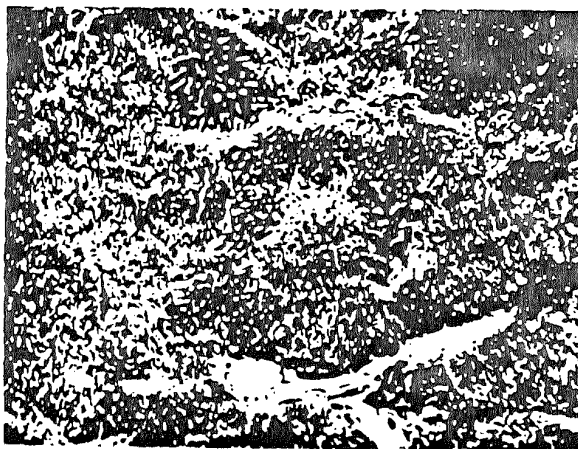


Fig. 2

liver cells which appeared swollen and more granular than normal. The cytoplasm of occasional cells contained small acidophilic hyaline droplets and there was a moderate excess of fat in the form of tiny vacuoles (fig. 6, plate VII). All microscopic abnormalities increased slightly during the second period (37 to 72 days) but no significant advance was detected between the 72nd and 130th day of exposure.

The livers of rats exposed for 105 days and then removed from exposure for a period of 2 months still showed cellular changes similar to those observed at the beginning of the recovery period. In a few of the specimens they appeared slightly less marked, indicating that some repair had taken place. In no instance was there evidence of increased damage.

Carbon tetrachloride and ethyl alcohol administered to rats exposed in the above manner for 143 days proved highly fatal, 9 out of 10 rats dying within the first 6 days, 6 within 72 hours. At autopsy the livers of these rats were yellow and mottled and they were increased in weight (16 to 44 per cent). Microscopic examination showed widepread central necrosis similar to that observed in early acute yellow atrophy of man (see

figs. 1 and 2, plate VII). The central one-half or two-thirds of each liver lobule showed complete liver cell necrosis and degeneration. Diffuse extravasation of erythrocytes had occurred and numerous polymorphonuclear and mononuclear leucocytes had invaded the necrotic areas. In the narrow periportal zones where the liver cells were less markedly damaged, there were large numbers of mitotic figures (fig. 2, plate VII).

(b) *Exposure by inhalation to low concentrations (average 1.77 mgms. per cu. m.) 8 hours daily.* Animals were subjected to these conditions for 143 days. Groups of animals sacrificed after 42, 77, 98, 119, and 143 days' exposure showed liver changes that were essentially like those seen in rats exposed 16 hours daily. The carbon tetrachloride and alcohol test was uniformly fatal and produced liver changes that were identical to those already described and illustrated (figs. 1 and 2, plate VII).

(c) *Exposure by inhalation to high concentrations (average 888 mgms. per cu. m.) 16 hours daily.* Pentachloro and hexachlorophthalenes when administered in this concentration proved highly toxic. Eighty animals were

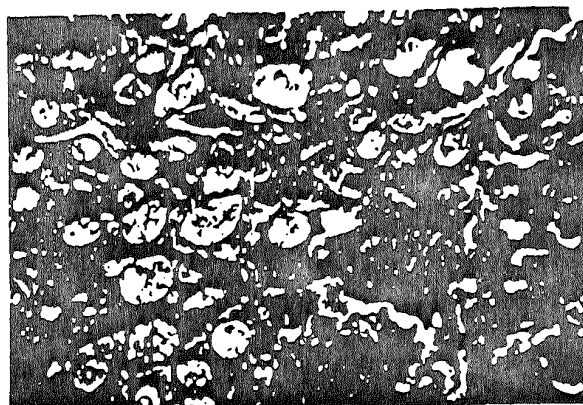


FIG. 1

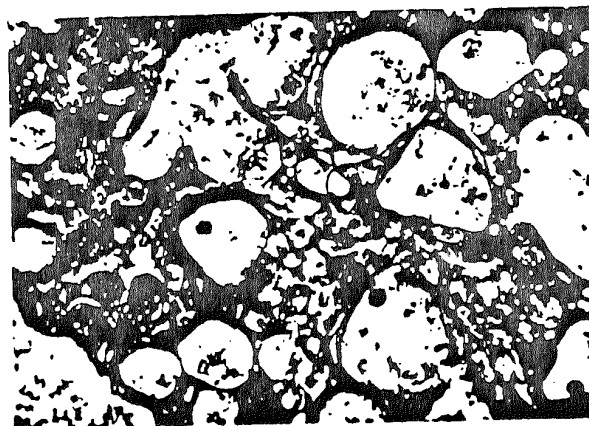


FIG. 2

PLATE VI

Fig. 1. Photomicrograph $\times 230$ of the liver of a rat exposed to pentachlorophthalene and hexachlorophthalene by the feeding of large daily doses for 72 days. The liver cells are swollen and markedly vacuolated. There is distortion of the liver cell columns. In frozen section preparations stained with Scharlach II, there was evidence of marked fatty degeneration. In addition to these changes, numerous oval round and irregular intercellular spaces are present throughout the liver lobules. Such spaces contain a granular serous precipitate, strands of

fibrous and occasional leucocytes. These spaces, similar to those found in the lungs, were due to dilatation of the bile capillaries and sinusoids.

Fig. 2. This photomicrograph $\times 216$, when compared with the above figure, demonstrates the similarity of the liver changes produced by the same compound administered in a different manner. In this instance the rat inhaled high concentrations of pentachlorophthalene and hexachlorophthalene for 38 days.

used, all of which lost weight and appetite. Four died by the end of the first month, 55 within 52 days. Most of these were markedly pumiced. The remainder were sacrificed for pathological examination or succumbed to the effects of carbon tetrachloride and alcohol. Only 8 animals survived the 52 day exposure. The majority of livers were moderately or markedly enlarged. They were all yellow and in addition the majority were markedly mottled. Occasionally they were granular and their cut surfaces showed minute spaces which became more prominent after fixation. After an 8 day exposure, the liver cells were markedly swollen and moderate fatty degeneration was present. This was most marked in the central areas although all portions of the liver lobules were affected. The extent of liver injury rapidly increased so that after 30 days' exposure, marked structural as well as cellular changes were apparent. The microscopic appearances were identical to those to be described in rats fed large doses of pentachlorophthalenes (figs. 1 and 2, plate VI) and in rats fed similar doses of 90 per cent pentachlorophthalenes plus 10 per cent chlorinated diphenyl (see fig. 1, plate III and figs. 1 and 2, plate VIII).

PLATE VII

Fig. 1. A low power photomicrograph $\times 15$ illustrating the marked degree of degeneration of liver tissue resulting from the administration of pentachlorophthalenes at a dose of 1.0 g. per kg. to rats which had been exposed previously by inhalation to those of low concentrations of pentachlorophthalenes for 30 days. This animal died within 24 hours after the administration of carbon tetrachloride.

Rats removed from exposure between the third and fifth week continued to die, the longest survival time being 35 days. Microscopic examination of the livers of these animals revealed no evidence of recovery.

Administration of carbon tetrachloride and alcohol again proved fatal to animals exposed in the above manner. The livers of such animals showed the characteristic massive necrosis previously described and illustrated.

(d) *Exposure by feeding*—When fed in doses of 3 gm. daily to a group of 10 rats, pentachlorophthalenes produced marked cellular and structural changes in the liver in a short period of time. All rats lost weight and appeared ill from the beginning. The longest survival time was 33 days. Although the liver edges were blunt and rounded, there were no significant weight variations. The livers were friable, yellow and mottled. Microscopically they showed marked swelling and vacuolization of the cells. There was also complete degeneration of scattered cells. Occasional mitotic figures were observed (see fig. 1, plate II). Suitable stains revealed very marked fatty degeneration. Occasional cells contained oval shaped or circular acid-

Similar degrees of necrosis resulted from the administration of carbon tetrachloride and alcohol to rats which had been exposed to a mixture of 90 per cent pentachlorophthalenes and 10 per cent chlorinated diphenyl.

Fig. 2. A higher power photomicrograph $\times 225$ of the liver illustrated in the above figure.

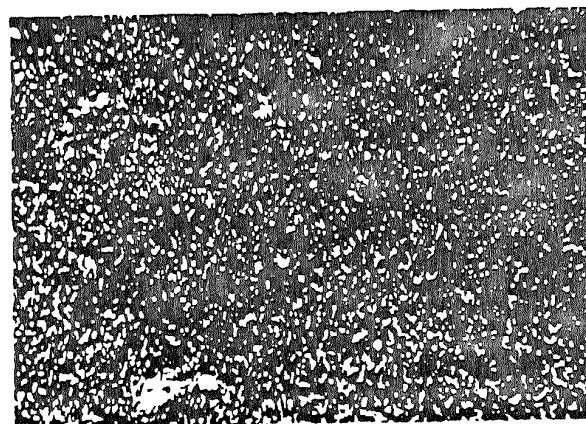


FIG. 1

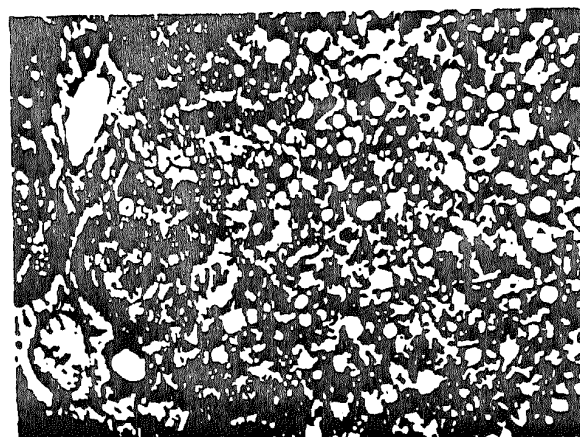


FIG. 2

ophilic hyaline inclusions. In addition to these cytological changes there were numerous varying sized intercellular spaces throughout the livers (see figs. 1 and 2, plate VI and fig. 1, plate III). These spaces were usually circular or oval in shape, although many of them ramified between cells in the liver columns or between liver cells and the sinusoidal endothelium in such a way as to suggest that they represented greatly dilated bile capillaries or canaliculi (fig. 1, plate III). They contained serous precipitate, a few strands of fibrin, and occasionally a few leucocytes. Rarely one observed one or two degenerating liver cells within these spaces. In some of the more markedly damaged livers, red blood corpuscles were also present. The above microscopic changes appeared identical to those observed in the livers of rats subjected to high concentrations of this compound by inhalation methods (figs. 1 and 2, plate VI). They were also similar to the changes that resulted from the feeding of another penta- and hexachloronaphthalene compound (figs. 5 and 6, plate I) and from the feeding of a mixture of penta- and hexachloronaphthalene (90 per cent) and chlorinated diphenyl (10 per cent).

When this preparation of penta- and hexachloronaphthalenes was fed in smaller amounts (0.5 gm. every second

day to a group of 4 rats) the above described liver changes occurred less rapidly and to a lesser degree (see fig. 5, plate II).

Compound E: Penta- and Hexachloronaphthalenes

Exposure by feeding. Feeding in doses of 1 gm. daily for 10 rats was continued with but one 4-day interruption. All 10 rats had died or had been sacrificed by the fifty-fifth day. These livers were friable and yellowish, and exhibited moderate to marked mottling. One animal was pouched of the 5 animals that were sacrificed, three showed enlargement of the liver (30 to 40 per cent), the remaining livers were normal in size. On microscopic examination the livers of rats exposed for 2 weeks showed marked cellular changes that appeared to be degenerative in nature. Although the liver cell injury was somewhat greater in the central portions of the liver lobules, no portion of the liver was spared. The liver cells showed varying degrees of swelling, increased granularity and vacuolization of the cytoplasm. Early injury was indicated by a massing of basophilic granules and small rod-shaped structures near the cell nuclei. The peripheral portions of such cells were acidophilic and vacuolated. Small triangular spaces were present between

PLATE VIII

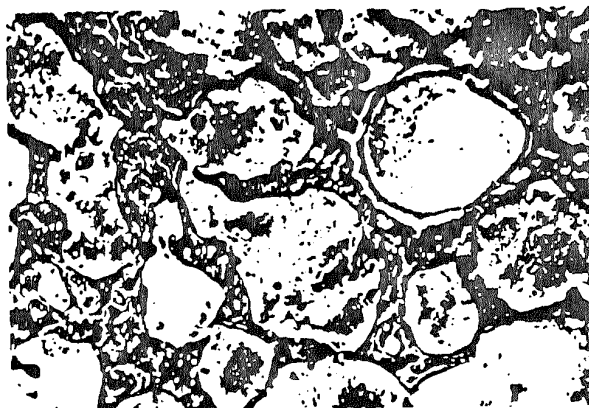


FIG. 1

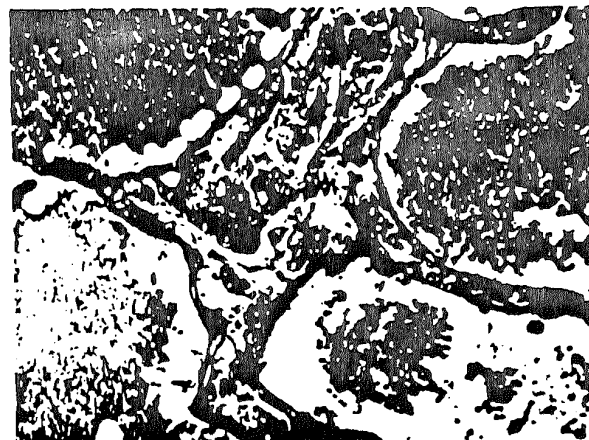


FIG. 2

PLATE VIII

FIG. 1. A photomicrograph $\times 95$ of the liver of a rat fed a mixture of 90% penta- and hexachloronaphthalenes and 10% chlorinated diphenyl in 3 gm. doses per 10 rats for 42 days. This rat survived for 23 days after feeding was stopped. A comparison of the liver of this rat with those of rats sacrificed earlier indicated that the liver changes had progressed considerably after administration of the compound had been terminated.

One should note the marked change in the liver architecture. The spaces like those previously illustrated (figs. 1 and 2, plate VI) are very large and the remaining liver tissue is compressed. The liver cells show marked fatty degeneration.

FIG. 2. A higher power photomicrograph $\times 295$ from another area in the liver illustrated in the above figure.

liver cells and between the columns of liver cells and the sinusoidal endothelium (see fig. 5, plate I). These were interpreted to be the early stages in the formation of large intercellular spaces (fig. 6, plate I) which subsequently developed and which were identical to the spaces described in the foregoing section. After longer exposure the liver cells showed very marked swelling and fatty infiltration. Occasional degenerating and regenerating cells were observed.

Compound F: 90 per cent Pentachloronaphthalene and 10 per cent Chlorinated Diphenyl

(a) *Exposure by inhalation.* Inhalation experiments were carried out on two groups of rats. The first of these groups of 80 animals were exposed to low concentrations (average 1.37 mgms. per cu. m.) 16 hours daily for 144 days. The second group, also comprised of 80 rats, was exposed to low concentrations (average 1.66 mgms. per cu. m.) 8 hours daily for 144 days. No evidence of illness was noted in the living animals in either group. In each experiment representative groups of animals were sacrificed after exposure periods corresponding to those used in low concentration inhalation experiments with trichloronaphthalenes and pentachloronaphthalenes.

Since there were no recognizable differences in the amount or type of liver change observed after similar exposure periods in these two groups of rats, the second group (exposed 8 hours daily) will not be discussed.

Macroscopically the livers were usually light yellow. After prolonged exposure, this abnormality was

more marked and in many instances mottling was present. There were no very significant alterations in the weights of livers although the majority were slightly swollen (average of 20 per cent increase in weight). Microscopically the livers showed constant changes after 37 days of exposure. These increased somewhat during the second period of 35 days. Longer exposure produced little if any increase in liver damage. The observed pathological changes consisted of swelling and rounding of liver cells accompanied by a definite increase in the prominence of the cytoplasmic granules. Hyaline droplets in the altered cytoplasm were a conspicuous feature (see fig. 3, plate III). Mitotic figures were present in abnormally large numbers. In an occasional liver, as was the case with similar exposure to both pentachloronaphthalenes alone and refined chlorinated diphenyl, there was a marked deposit of finely divided granular and refractile yellow or yellow brown pigment. This was present within liver and Kupfer cells. Larger amounts were found in the central portions of the liver lobules. It failed to stain in a manner characteristic of either hemosiderin or hemolysin.

The livers of rats removed from exposure for 2 months after an exposure period of 105 days were similar to those of animals sacrificed at the end of 105 days' exposure. There was, however, no evidence of any increase in the pathological changes.

The administration of carbon tetrachloride and alcohol was almost uniformly fatal. Macroscopic examination of livers of these rats revealed marked enlargement (approximately

60 per cent). Such livers were yellow and mottled. Again there was extensive and widespread liver necrosis on microscopic examination (see figs. 1 and 2, plate VII).

(b) *Exposure by feeding.*—When fed in large doses (3 gm. daily for 10 rats) this compound proved to be very toxic. All animals appeared ill and feeding of the compound was stopped after 12 days. Despite this the rats continued to die, the last dying on the 28th day. Subsequent microscopic examination revealed that the liver changes had continued to progress after feeding was stopped. Macroscopically, the majority of the livers were enlarged, the largest showing an increase in weight of 118 per cent; the average increase was approximately 40 per cent. All livers were yellow, friable, and many of them were markedly mottled. Subsequent microscopic examination indicated that much of the mottling was due to hemorrhage into spaces within the liver tissue. In addition, the external and cut surfaces of the livers appeared slightly pitted or granular. The microscopic changes observed in the livers of the animals autopsied after 10 to 14 days' exposure consisted of moderate to marked swelling of liver cells accompanied by marked fatty vacuolization. (Occasional liver cells showed additional signs of injury such as nuclear degeneration and polymorphonuclear leucocytic invasion. Scattered circular or oval shaped intercellular spaces (fig. 1, plate III) like those previously described in experiments where pentachloronaphthalenes were employed (figs. 1 and 2, plate VI) were observed. In

the less markedly damaged livers, such spaces contained serous precipitate, strands of fibrin, and small numbers of leucocytes. In rats surviving 18 to 26 days, these spaces were much larger and often had attained the size of a normal liver lobule (see figs. 1 and 2, plate VIII). In these more severely damaged specimens, extensive hemorrhage had occurred (fig. 2, plate VIII). In sections stained with P.T.A.H. these spaces were seen to be bounded by flattened liver cells which showed sharp cell borders. Liver cells between such spaces were distorted, swollen, and showed marked fatty degeneration. In many of these livers there was a slight increase in connective tissue between the remaining cords of liver cells and around recognizable portal areas. Proliferative changes were occasionally observed in the bile ducts. This was indicated by increased numbers of ducts in certain areas and by mitotic figures in the bile duct epithelial cells. No recognizable dilatation of medium sized or large bile ducts was present. In many sections the architecture of the liver was so completely altered that it was difficult to recognize the portal areas. In all sections there was evidence of fatty and hyaline degeneration of the remaining liver cells. Slight polymorphonuclear leucocytic infiltration was present. This was somewhat more marked in the periportal areas.

Feeding of this mixture of pentachloronaphthalenes and chlorinated diphenyl in smaller doses (0.5 gm. every second day for 4 rats) resulted in liver damage that developed less rapidly. The rats were sacrificed after exposure periods of

21, 34, 58, and 81 days. Their livers were yellow, increased in size and showed slight mottling. Microscopically, the chief abnormality observed was marked fatty vacuolization of liver cells. No intercellular spaces like those described above were present.

Compound G. Chlorinated Diphenyl

(a) Exposure by inhalation. Exposure by inhalation methods to low concentrations of chlorinated diphenyl was carried out on two groups of animals, each comprised of 80 rats, in a manner identical to that described in the foregoing sections. In the first group a 16 hour daily exposure, with an average concentration of 0.57 mgms per cu. m., was employed. The second group of animals was exposed 8 hours daily to an average concentration of 0.93 mgms per cu. m. Neither group of rats appeared ill. Representative animals were sacrificed in groups of 3 to 21 rats, after varying periods of exposure. In the first experiment, the animals were sacrificed after 37, 72, 105, and 134 days of exposure, in the second experiment after 42, 77, 98, 119, and 143 days. The livers were pale or slightly yellow and somewhat mottled. There were no constant variations in the weights of the livers. The microscopic findings in the two groups indicated that the different exposures had resulted in the same degree of liver injury. The pigmentation of the liver and Kupffer cells was somewhat greater in animals exposed 8 hours daily. In both experimental groups the carbon tetrachloride and alcohol test was highly fatal and led to the same extensive central necrosis of liver tissue that was observed in rats exposed to

the toxic chlorinated naphthalene compounds (figs. 1 and 2, plate VII). The microscopic changes resulting from exposure to low concentration of chlorinated diphenyl alone were similar to those resulting from inhalation of penta- and hexachloronaphthalenes, plus 10 per cent chlorinated diphenyl. The most conspicuous change was hyaline degeneration (fig. 6, plate III) although swelling of liver cells, increased prominence of cytoplasmic granules and increased vacuolization were present. Mitotic figures were present in increased numbers. No microscopic evidence of recovery was found in the livers of rats exposed for 105 days and then removed from exposure for a period of 2 months.

(b) Exposure by feeding. When fed in large daily doses (3 gm. for groups of 10 rats), chlorinated diphenyl proved highly toxic. Feeding was discontinued after 6 days. Seven rats died within the first 8 days. The 3 remaining rats gained in weight after the feeding was stopped and were finally sacrificed. The last animal was sacrificed 63 days after feeding was begun. The majority of livers showed a moderate increase in weight. Microscopically there was evidence that liver injury had occurred within the first few days. The cells were swollen, at times sufficiently to obscure the sinusoids. The cytoplasm of the liver cells was acidophilic in staining quality and contained small vacuoles. Small eosin-stained hyaline granules and globules were present. Mitotic figures were numerous (see fig. 4, plate III). There was little inflammatory cell infiltration and no structural changes were observed.

When smaller doses (0.5 gm. every second day) of this compound were administered to 10 rats, the first death occurred after 9 days. Two additional deaths occurred after 12 days and two more by the end of the fifth week. The remaining animals were sacrificed between the 40th and the 100th day. The livers were enlarged, the average increase in liver weight being 33 per cent. They were pale and slightly mottled. The most outstanding microscopic abnormality was the presence of large numbers of varying sized acidophilic hyaline globules in the cytoplasm (see fig. 5, plate III). These hyaline globules, although larger and more numerous, appeared identical to those encountered in the livers of rats exposed to other chlorinated diphenyl preparations. The liver cells were markedly swollen and showed fatty vacuolization. There was also increased regenerative activity. No structural changes were observed in any of the livers.

Discussion

Published reports concerned with the possible systemic effects of chlorinated naphthalenes have been few in number. Lehmann (3) in 1919 reported that animals exposed to chlorinated naphthalenes by inhalation or feeding refused to eat and at death showed "peculiar" lesions in the liver. In 1936, Flinn and Jarvik (4) reported the results observed following the daily subcutaneous injections into 30 rabbits of a mixture of tri- and tetrachloronaphthalenes (30 mgm.) of certain chlorinated naphthalene compounds dissolved in paraffin oil. The rabbits receiving a mixture of tri- and tetrachloronaphthalenes lived and when sacrificed, after a 2 months'

exposure, no pathological changes attributable to the compound injected were demonstrable. The mixtures of tri- and pentachloronaphthalenes and of penta- and hexachloronaphthalenes proved highly fatal and degeneration of liver tissue was observed. From their findings Flinn and Jarvik concluded that certain chlorinated naphthalenes or impurities contained in them are capable of producing yellow atrophy of the liver in the rabbit. It is noteworthy, in the light of the present experiments, that the compound of lowest chlorination (a mixture of tri- and tetrachloronaphthalenes) had no apparent effect on the well being of the animals and did not produce demonstrable changes in the liver although injected daily for a period of 2 months.

The present experiments demonstrate that chlorinated naphthalene compounds and chlorinated diphenyl are capable of producing marked liver damage in the white rat without demonstrable microscopic changes appearing in the other organs. Furthermore, the characteristics of the liver lesions resulting from comparable amounts of any given compound are the same, regardless of the method of administration (inhalation, feeding, or subcutaneous injection).

An attempt has been made throughout this study to grade the toxicity of each of the several compounds tested in accordance with the severity of the liver damage produced. Although none of these compounds appeared to produce similar degrees of liver injury, the toxicity of each compound seemed directly related to its degree of chlorination. Thus, a mixture of trichloronaphthalenes (chlorine content 49.4

per cent) proved to be the least toxic of any of the compounds tested, whereas chlorinated diphenyl (chlorine content 65.0 per cent) was highly toxic even in very low concentrations.

Trichloronaphthalenes, when compared to other naphthalene compounds of higher degree of chlorination, were relatively innocuous. Even after prolonged feeding of large amounts of this material, the rats showed no evidence of ill health. After 2 months' exposure, microscopic examination of the livers revealed only slight to moderate degrees of fatty infiltration and degeneration of liver cells (plate II, figs. 1 and 2). These changes were slight when compared to those resulting from short exposures to compounds of higher chlorination. Exposure by inhalation to low concentrations of trichloronaphthalenes produced liver cell alterations that were never more than minimal (see fig. 3, plate II). Such animals withstood additional liver injury from carbon tetrachloride and alcohol in a manner indistinguishable from that of the normal animal. Somewhat greater liver injury resulted from inhalation when the air concentration of this compound was increased more than eight times that used in the previous experiment but again the liver changes were slight compared to those produced by comparable exposure to the more highly chlorinated compounds.

The mixture of tetra- and pentachloronaphthalenes employed in these experiments appeared to be considerably more toxic than was the mixture of trichloronaphthalenes. Animals fed small doses of the former of these preparations fell ill and died or were sacrificed by the 63rd day. Their livers showed extensive fatty infiltra-

tion and fatty degeneration (figs. 3 and 4, plate I).

Feeding of tetra- and pentachloronaphthalenes in combination with chlorinated diphenyl resulted in pronounced liver changes. These livers had increased in weight (average 71 per cent). Microscopic examination revealed a peculiar type of hyaline degeneration involving practically every liver cell (see figs. 1 and 2, plate I). This type of cell degeneration was more marked and occurred earlier after exposure to preparations containing chlorinated diphenyl than to any other compounds tested. Furthermore, it was most marked in the livers of animals exposed to refined chlorinated diphenyl (figs. 4, 5, and 6, plate III).

Comparable exposure of rats to penta- and hexachloronaphthalenes (Compound D), penta- and hexachloronaphthalenes (Compound E), and penta- and hexachloronaphthalenes (90 per cent) plus chlorinated diphenyl (10 per cent) (Compound F), resulted in liver changes having few detectable differences.

The morphological changes were slightly greater in the livers of rats exposed to the last named compound. In these instances the hyaline degeneration of the cell cytoplasm was more marked. Although rats inhaling low concentrations of compounds D and F showed no demonstrable signs of ill health, microscopic examination of their livers revealed marked liver cell injury (fig. 6, plate II, and fig. 3, plate III). These lesions were still demonstrable after a 2 months' recovery period. Further evidence that liver damage had resulted from inhalation in low concentrations of these compounds was obtained from the carbon tetra-

chloride and alcohol test. Such tests were almost uniformly fatal and resulted in widespread liver necrosis (figs. 1 and 2, plate VII). These three compounds proved to be very toxic and resulted in a most unusual type of liver change when administered in large amounts by feeding (see fig. 1, plate III, fig. 1, plate VI, and figs. 1 and 2, plate VIII). Identical changes were observed following inhalation in high concentrations of compound D (see fig. 2, plate VI). In such livers the most conspicuous feature was the presence of large intercellular spaces. Examination of routine sections, supplemented by specially stained preparations indicated that these spaces had resulted from dilatation of bile canaliculi. The reason for such dilatation was not apparent. There was no corresponding dilatation of the large and small bile ducts at the periphery of the liver lobules and no inspissated bile was ever observed. The organically combined chloride in the livers of these rats was no greater than that observed in the normal animal. Although serious liver damage results from exposure to high concentrations of these compounds, the resulting changes are most unusual and do not resemble those of acute yellow atrophy on microscopic examination.

Of the various chlorinated hydrocarbons tested, chlorinated diphenyl gave evidence of being the most toxic. When administered by inhalation in very low concentrations (average 0.67 to 0.99 mgms. per cu. m.) liver cell changes were very pronounced after the first exposure period. The most striking change was the hyalinization of the cell cytoplasm (see fig. 6, plate III). Such cellular alterations were

essentially unchanged after a 2 month recovery period. In these animals small sublethal doses of carbon tetrachloride and alcohol uniformly produced extensive liver necrosis and was highly fatal to them (figs. 1 and 2, plate VII). Chlorinated diphenyl fed in small doses produced similar but more marked liver injury (see fig. 6, plate III). In large doses this compound was highly fatal. The liver changes in animals dying after short exposures were inconspicuous and consisted mainly of swelling of cells and active regeneration (see fig. 4, plate III). However, animals removed from exposure before being fatally poisoned, subsequently developed hyaline degeneration of liver cells similar to that produced by prolonged administration of small doses of this compound.

Thus the results of the present study, as well as certain field studies that have been made (1) suggest that the solution of the industrial hazard involved is dependent largely on a reduction of the air concentration of these compounds to a level that will not produce liver damage. The present experiments indicate that lower air concentrations must be obtained in the case of the more highly chlorinated naphthalene compounds and chlorinated diphenyl than for trichloronaphthalenes if a safe environment for workmen is to be assured. Because of the pronounced toxic effect of small doses of carbon tetrachloride on the livers of animals already injured by exposure to chlorinated naphthalenes and chlorinated diphenyl, its use as a solvent for these compounds would appear to be very hazardous.

Although this investigation was designed to determine what systemic

effects, if any, would be produced in experimental animals by a group of chlorinated hydrocarbons, the pathological changes produced have been of such a nature as to justify further comment. Exposure of rats to highly chlorinated naphthalene compounds and chlorinated diphenyl results in a type of liver injury differing considerably from the liver lesions resulting from carbon tetrachloride (5, 6) and chloroform poisoning (7). The changes resulting from exposure to the compounds employed in this study involve all portions of the liver lobule. This is true despite the fact that the initial morphological changes are more pronounced in the liver cells nearest the hepatic veins. The changes observed are degenerative in nature. The successive degenerative changes were cloudy swelling, fatty infiltration and fatty degeneration and finally, the complete disintegration of the cell. Degeneration characterized by the accumulation of large amounts of acidophilic hyaline material was also observed. This feature was much more marked in the livers of animals exposed to the compounds containing chlorinated diphenyl. It is of interest that widespread degenerative changes of all liver cells can exist for several months without producing any evidence of ill health in the animals or without causing important structural changes in their livers. Such findings suggest that the alterations take place slowly and that the scattered necrotic cells are efficiently removed and replaced by new cells which also become injured. There was no observable morphological evidence to indicate that the regenerating liver cells had acquired an increased resistance to the injurious effects of the compound

being used (8). The fact that liver cell changes were still present after a 2 month period during which the animals were removed from exposure to the more highly chlorinated compounds is further evidence that this type of injury is persistent and only slowly recovered from.

The administration in high concentrations of two compounds of intermediate degrees of chlorination resulted in marked structural changes in addition to marked degenerative changes in liver cells. The most conspicuous change was the occurrence of progressively enlarging intercellular spaces. These were interpreted as markedly dilated bile canaliculi. We are unaware of such changes having been previously described.

From the above it is apparent that exposure to highly chlorinated naphthalenes and chlorinated diphenyl not only results in liver changes having marked differences from those caused by other well known toxic agents, but also that the characteristics of the liver changes produced can be markedly altered quantitatively and qualitatively by varying the degree of exposure. For these reasons one would seem justified in suggesting that detailed studies on animals in which the types of liver injury observed in these experiments had been produced might further our understanding of liver cell function.

SUMMARY

1. Studies concerning the effect upon white rats of a group of chlorinated naphthalene compounds and chlorinated diphenyl are reported. This investigation was undertaken because of the recent occurrence of 3 fatal cases of jaundice in men work-

ing in industrial plants where these compounds were being used. The compounds tested were selected as representative of a certain range of chlorination and because of their relative industrial importance.

2. Four of the compounds tested were administered by inhalation. The air concentration and exposure periods were varied. All compounds were tested by feeding in large and small amounts. In addition three of them were tested on small numbers of animals by subcutaneous injections. The effects of any given compound did not appear to be influenced by the method of administration. However, marked quantitative and qualitative differences in the effects of each of several compounds resulted from varying the air concentrations in inhalation experiments and the dosage employed in feeding experiments.

3. Macroscopic and microscopic examination of the tissues of rats exposed to these compounds indicated that the injurious effects are manifested solely in the liver. Blood examination during life revealed no significant abnormalities.

4. The liver changes resulting from exposure to the chlorinated hydrocarbons employed have been described and illustrated. As indicated by their ability to injure liver tissue the toxicity of these compounds increases with increasing degrees of chlorination. Thus a mixture of trichloronaphthalene is relatively innocuous in low concentrations and when compared with compounds of higher chlorination, its toxic properties even in higher concentrations are relatively slight. Chlorinated diphenyl appears to be the most injurious compound of all those tested.

5. Administration of small sublethal doses of carbon tetrachloride and ethyl alcohol to rats whose livers have already been injured by the compounds under consideration is highly fatal and produces massive necrosis of the liver.

6. The significance of this study in relation to the industrial problem involved is discussed.

7. It is suggested that the types of liver injury observed in this study might be employed in experiments designed to study liver cell function.

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*Liver Disease from
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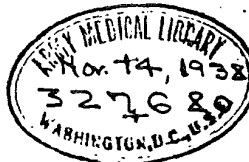
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LIVER LESIONS CAUSED BY CHLORINATED NAPHTHA- LENE¹

By

FREDERICK B. FLINN AND NORMAN E. JARVIK

(Received for publication July 21, 1937)

In this country nine cases of yellow atrophy of the liver have occurred in shops in which chlorinated naphthalenes are being used to impregnate wire and other electrical equipment. The total number of employees exposed in the three plants was approximately 250. The fact that three of these cases have occurred, in each of three different plants, all widely separated and under different management but using the same type of material, indicates the possibility of a common etiological factor. The average time of exposure varied from 4 to 6 months.

Yellow atrophy of the liver is a rare disease. Osler (1) reported three cases among 28,000 patients examined. The records of 5,000 autopsies at the Presbyterian Hospital in New York, show three acute cases and fifteen sub-acute cases. Up to 1913, Umber (2), in Germany, had not seen a single case among 84 autopsies performed. In 1920, he reported two cases in 200 autopsies, and in 1921, eleven in 149 autopsies. Seyfarth (3), also of Germany, had had only one case up to 1915; but between 1915 and 1921 he found twenty-nine cases of yellow atrophy. The increase in the number in Germany appeared during the World War, at which time the use of chlorinated naphthalenes also increased. The majority of the cases, however, occurred in women during the middle or latter half of pregnancy, and no record of exposure to chlorinated naphthalenes in these cases has been reported.

We have been unable to find reports in the literature of any case of yellow atrophy occurring among the workers exposed to chlorinated naphthalenes either in this country or Europe. The hazard commonly recognized has been a cutaneous one, the lesion being in the

¹ From the Institute of Public Health, Columbia University, New York, N. Y.
We want to thank Dr. William C. Von Glahn for his assistance in studying the pathology of the animals autopsied.

form of an acne comedon. One hundred and two cases of dermatitis were found among 124 workers in Pennsylvania and a few mild cases in another plant. These cutaneous lesions have been reported by Wauer (4), Koelsch (5) and Teleky (6) under the name of "Perna Disease" which had resulted from exposure to a perchloronaphthalene. Perna wax is, in general, a mixture of chlorination products of naphthalene. It is yellow to dark brown in color, soluble in fats, contains 44 to 45 per cent of chlorine and sublimes at 130° C. Teleky showed that the cause of cutaneous injuries was definitely due to the easily volatile chlorine compounds contained in "Perna" to the extent of only 5 per cent. Lehmann (7) experimented with animals and found that this chemical produced lesions of the liver which resembled acute yellow atrophy. Observers in France and Germany agree that in spite of the claim of individual susceptibility, 50 per cent of the workers showed cutaneous lesions.

The failure to find reports of any cases of atrophy among industrial workers exposed to chlorinated naphthalene may be explained by the history of similar industrial diseases. The medical profession at the beginning did not recognize the connection of the pathologic condition with an industrial exposure. Frequently, correct diagnoses are not made when the first cases make their appearance. In this instance the first cases in each locality were diagnosed as catarrhal jaundice in spite of the fact that catarrhal jaundice is rarely fatal. Post mortem examination which revealed the correct nature of the disease was not made until the second or third case appeared.

In the factory to which we were called to determine the etiological agent we found three types of chlorinated naphthalenes in use. They are sold under the name of halowaxes and, in this paper, will be designated as compounds A, B, and C. Compound C was introduced into the factory at the beginning of the 6-month period during which three cases occurred.

Compound A is a mixture of tri- and tetrachloronaphthalene. Compound B is a mixture of tetra- and pentachloronaphthalene and may contain some trichloronaphthalene. Compound C is a mixture of penta- and hexa-chloronaphthalene which is plasticized with a relatively small percentage of asphalt.

A boils between 600 and 650° F.; B boils between 615 and 655° F.; C boils between 680 and 730° F.

We determined the chlorine content and the melting point of each of these materials. The sublimate given off by B at 192° C. and that given off by C at 172° C. were collected and analyzed.

Compound	Chlorine percentage	Melting point
A	47.9	89 - 90° C.
B	49.8	117.5-122° C.
C	47.9	123 -132° C.
B sublimate	35.3	87 - 89° C.
C sublimate	53.1	117 -123° C.

While the circumstantial evidence pointed to the chlorinated naphthalenes as the etiological agent, we included in our study other materials that were being used in the plant during the period in which the disease developed. These substances were found not to produce the liver lesions. We therefore concentrated on the naphthalenes and used them in amounts that would be certain to produce results if they were at all toxic. In the preliminary part of the study we were not interested in determining the minimum toxic dose.

The first symptoms noted by the affected employee were a "run-down" condition and jaundice. As the jaundice increased there appeared an increase in general malaise, anorexia, attacks of dizziness, loss of weight and a tendency to vomit. Severe abdominal pains across the upper quadrant were noticed. The blood picture was normal except for a low blood sugar which in several cases was around 68 mgm. per 100 cc.

A typical gross autopsy finding showed an extensive necrosis, fibrosis and regeneration of the liver, acute enterocolitis with edema, fibrosis of the pancreas, acute pancreatitis, jaundice, ascites, edema of lower extremities, and subpleural hemorrhages.

EXPERIMENTAL

It has been our experience that the rabbit is the best laboratory animal for the study of liver lesions. Rabbits weighing approximately 2 kilos were used in the study. We also included a few rats and guinea pigs in these experiments, which brought the total number of animals used up to two hundred.

Dose. Fortunately for our investigation the State Division of Occupational Hygiene of Massachusetts had made a test of the air in the plant we were investigating when the last case appeared. Their findings are as follows: Rolling Department, 1 to 2 mgm. of wax per cubic meter of air; Soldering Department, 1 to 2 mgm. of wax per cubic meter of air; Impregnating Department, 11 to 20 mgm. of wax per cubic meter of air.

Our observations in this plant and laboratory experiments made

us feel that even 20 mgm. of wax per cubic meter of air in some localities may be low. The chlornaphthalene crystallizes readily out of the air within very short distances from the tank in which it is being heated. For this reason we felt that it was very difficult to determine the exact concentration to which a man might be exposed. Taking into consideration that a man breathes from 4.5 to 5 cubic meters of air during his working hours, we selected 120 mgm. as the dose a man might possibly inhale in a day's work. There were no available data at that time to aid us in determining the dose which might have caused liver lesions in man. We wish to emphasize that this arbitrary dose of 120 mgm. was selected only as a starting point for our work.

The naphthalene was dissolved in paraffin oil heated up to the point where the chlornaphthalene went into solution. We have determined from other work that the oil does not produce pathologic changes which would interfere in this work. For the reasons stated we chose the method of subcutaneous injection rather than the inhalation method for exposure because we felt we could, in this way, control the intake absolutely. We were further urged to use the subcutaneous method because of our experience with tri-cresyl-phosphate, which causes the so-called "ginger paralysis" in man. It was found that when the rat, guinea pig, cat, dog, and monkey were fed tri-cresyl-phosphate paralysis did not occur; but if they were given subcutaneous injections the lesion appeared. On the other hand, calves, rabbits or chickens became paralyzed when fed (the chemical) by mouth. The subcutaneous method was effective in all the species. The work of Lehmann (7) indicated that the liver was injured whether the animal inhaled chlornaphthalene or was fed the compound. Our own experimental work confirmed this.

We have found in our studies that the rabbit frequently requires the same dose as man to produce similar results. The toxic dose of a substance per kilo of body weight is frequently many times higher for the rat than for the rabbit. We found that 120 mgm. of the chlorinated naphthalene killed the rabbit too quickly to produce the final picture of acute atrophy of the liver. The dose was reduced to 30 mgm. Our observation showed that even this smaller dose was too large to produce the final picture of atrophy in rabbits. These doses brought out very definitely the toxicity of the material, at least for rabbits, which was the main object of this preliminary study. One hundred per cent fatality occurred in the groups of animals receiving chlorinated naphthalenes *B* and *C*, and sublimate from *C*.

On the other hand, no fatalities occurred in the groups receiving the compound *A* and sublimate *B*.

Our procedure was as follows: groups of five rabbits were used for each compound. They received daily injections until they died or were sacrificed. Then the work was repeated with the same number of animals. Aside from loss of weight, we were unable to detect any symptoms among the injected rabbits in the groups receiving compounds *B* and *C* and sublimate *C*. The control rabbits receiving paraffin oil, compound *A* and sublimate from *B*, gained weight. The blood picture showed no change in the red, white, or differential count. During the experiments the rabbits were fed the usual diet of water, hay, oats, and cabbage or lettuce.

Our results can be summarized as follows:

Chlornaphthalene:

- A*. All animals receiving either 120 or 30 mgm. daily remained alive until they were killed for post mortem examination at the end of two months.
- B*. The first death occurred in the group receiving 120 mgm. at the end of 12 days. The same holds true for the group receiving 30 mgm. None survived beyond 15 days.
- C*. The group receiving 120 mgm. daily were all dead by the end of 15 days. The first death occurred at the end of 9 days. In the group receiving 30 mgm. the first rabbit died in 12 days. The last of the group died on the twenty-sixth day.
- B Sublimate*. No deaths occurred in the groups receiving 120 mgm. or 30 mgm. They were sacrificed for autopsy at the end of 2 months.
- C Sublimate*. This group received 30 mgm. daily. The first rabbit died at the end of the ninth day and the last one on the fourteenth day.

We are giving two typical autopsy findings on the rabbits which received daily doses of 30 mgm. of compound *C*. Rabbits receiving compound *B*, *C* or *C* sublimate showed very much the same picture—perhaps in some cases not so far advanced. Each liver showed the yellow areas and necrosis.

Rabbit no. 533. Dosage 25 injections of 30 mgm. of Compound *C*. Died on the twenty-fifth day. Liver: Dark red with many opaque yellow areas; capsule smooth; cuts without resistance; cut surface smooth. In many parts of the organ there are yellow, opaque areas with dull surface, which involves groups of lobules. In between, the liver parenchyma is red in places and the lobulation is clearly visible. In other places, the lobulation cannot be seen, the parenchyma has ap-

parently disappeared and the stroma is collapsed; these portions are greyish red. The gall bladder is distended. Lungs: Many dark, red areas in all lobes. Cut surface moist; in the peritoneal cavity a moderate excess of bloody fluid is seen.

The presence of bloody or yellow fluid in the peritoneal cavity was a common finding in our rabbits. It was observed in human cases.

Histological findings: Heart: A very few phagocytes containing yellow pigment are found near arterioles. Lung: Capillary congestion and edema. Spleen: Some post mortem autolysis; phagocytes containing pigment in pulp. Adrenal: Normal. Kidney: In epithelial cells of the convoluted tubules there is a finely granular, yellow pigment. Liver: Presents a striking change. Beneath the capsule on one of the sections is a wide zone of necrosis in which portal areas are surrounded by a single row of well preserved cells, or none at all. In other places in the section efferent veins are surrounded by wide zones in which liver cells have undergone necrosis. There are numerous other places where liver cells have completely disappeared and only the collapsed stroma remains. In many places calcification of the stroma is well advanced and larger masses of calcium are frequently seen. Multinucleated giant cells of huge dimensions are often clustered about the masses of calcium or are found in the areas of collapsed stroma. Liver cells remain but regular normal architecture is not retained. Rounded groups of liver cells are numerous, or only narrow bands of these cells surround portal areas. The liver cells also present a peculiar arrangement in many places. A double column of the cells will open leaving a considerable space surrounded by the cells. The space contains granular precipitate or large plugs of bile. One gets the impression that these spaces may be hugely distended bile canaliculi, because in some instances they contain bile. Necrosis of individual liver cells is also seen. Cirrhosis negative.

Iron stain: Heart: Pigment stains red. Lung: No pigment. Spleen: Pigment does not stain well, perhaps because of autolysis. Some granules faintly blue, others yellow-red. Kidney: Pigment in moderate amount; red in color. Liver: Pigment red.

Rabbit no. 534. Daily dose of 30 mgm. of C for 21 days. Heart: Right ventricle dilated and filled with blood; otherwise normal. Lungs: Pink, air-containing and normal. Spleen: Soft, congested, but not enlarged. Pancreas: Appears larger than usual, otherwise normal. Liver: Capsule smooth and transparent except where there are large areas of yellow discoloration with irregular outlines. On section the yellow areas extend into the liver substance for a few mm., but almost everywhere are near, or in contact with, the surface. Where these yellow areas of necrosis are most discrete they seem to be in the portal areas. Elsewhere the portal areas are yellowish gray, the efferent vein regions red and the intervening parenchyma reddish brown. The lobulation, except in the necrotic yellow zone, is distinct and regular; no gross cirrhosis. It presents a very startling picture. Adrenals, kidney and stomach appear normal.

Histological findings: Heart: Negative. Lung: Negative except for one small calcified mass, probably a thrombus. Spleen: Malpighian bodies large; sinuses wide and full of blood; few phagocytes with pigment. Kidney: Normal. Liver: Extensive necrosis involving all liver cells of lobule or leaving a narrow zone about the portal areas. Rarely, the necrosis is more focal. Where liver

cells remain they often have compact, blue-staining areas in the cytoplasm. In this section, as in rabbit no. 533, there are wide spaces bordered by liver cells and at times containing large plugs of bile. It can be clearly seen that these spaces are distended bile canaliculi. Calcification is present in many of the areas of necrosis, stroma as well as liver cells. Large foreign body giant cells are found about the calcium. Some of the liver cells contain large droplets of fat, but total amount of fat is not great. Many liver cells contain bile. Cirrhosis negative; no proliferation of bile ducts.

Iron stain: Heart: No pigment. Lung: No pigment. Spleen: Small amount of blue pigment. Liver: Pigment red. Kidney: Very small amount of fine, red-staining pigment in epithelial cells of convoluted tubules.

For the purpose of determining the effects of smaller amounts of chlorinated naphthalenes we fed guinea pigs 5 mgm. per kilo of compound B for from 2 to 5 months. The pigs showed no clinical symptoms before death. The autopsies presented very much the same type of lesions as in the rabbits which had been receiving the large dose by injection. The liver surfaces were pebbled, and yellow areas beneath the capsule and deeper into the organ and areas of necrosis involving entire lobules were common.

A group of rabbits was injected with 2 mgm. of compound B per kilo of body weight. After a period of time sores began to appear in the skin. Histological section of these sores showed them to be areas of multiple, small necroses. As they only occurred in the chlornaphthalene animals we feel that this condition is due to the exposure.

The rabbits were killed after 7 months and autopsied. The picture of the liver showed the same type of necroses and yellow areas.

Our findings indicate that this small exposure does affect the liver of the rabbit although in these cases it will take a longer period of time to produce the lesions obtained by shorter exposure to the larger amount.

DISCUSSION

From the facts presented one arrives at the conclusion that certain of the commercial, chlorinated naphthalenes are toxic if they are absorbed by the body. This conclusion is warranted by the following facts: (1) there was 100 per cent fatality among the animals receiving injections of compounds B and C and sublimate C; (2) the livers of the animals receiving the injections showed the early stages of acute atrophy of the liver; (3) in spite of the rarity of this disease there occurred three human cases of yellow atrophy of the liver in each of three widely separated plants all using the same type of material.

Whether the toxic material is the chlorinated naphthalene itself, or some impurity contained in the commercial product, is the subject of further study. However, one may take into consideration the following facts. Several addition compounds of naphthalene are known. They are probably the intermediate product of naphthalene, such as penta- and hexachloronaphthalene. One would expect the chlorine addition products of naphthalene to be relatively unstable. As a matter of fact, the simplest one, naphthalene dichloride, slowly evolves HCl at temperatures as low as 40° to 50° C., giving the relatively stable monochloronaphthalene, a true chlorine substitution product. The higher chlorine addition products, and the compounds containing both the substituted and added chlorine which may be present as impurities, are more stable than naphthalene dichloride but, under certain conditions, they will give up HCl to form true chlorine substitution products.

This is only a working hypothesis. However, the observation can be explained by assuming the presence of a trace of these relatively unstable addition products, which may be absorbed by the skin and there give off HCl but which, upon suitable heating, would be partly or completely destroyed so that the vapors would be relatively less toxic. This may be the explanation in the case of compound B. In the case of compound C one must conclude either that the toxic impurities, if any are present, are not destroyed by simple heating or that this particular polychloronaphthalene is toxic *per se*. It is recognized that batches of chemically pure naphthalene will chlorinate in different ways at times.

SUMMARY

1. A 100 per cent fatality occurred when rabbits were injected subcutaneously with chlorinated naphthalenes B and C and the fumes of C. Guinea pigs fed by mouth gave the same result.

2. The liver lesions are those found in the early stages of yellow atrophy of the liver. After a longer exposure, small doses will cause lesions similar to those produced by the larger doses.

3. The cases of yellow atrophy of the liver among the employees using these chlorinated naphthalenes are probably due to an exposure to some chlorinated naphthalene.

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(314) OXford 4-1000

March 3, 1969

On February 24, the San Francisco Chronicle carried a major feature about "a menacing new pollutant" found in the San Francisco Bay area. The article was based on marine life research carried out by Dr. Robert Risebrough of the University of California. It stated that residues of polychlorinated biphenyl (PCB) were killing certain marine birds and posed a long-term threat to humans.

This story has caused considerable comment on the West Coast and its claims may be repeated elsewhere. As you know, Monsanto manufactures polychlorinated biphenyl and markets it under our Aroclor trade name. We, therefore, would like to present some additional facts.

The work done by Dr. Risebrough dates back to earlier research by other scientists to analyze the amount of pesticides in wildlife, soil and water. The initial pesticide research was extremely difficult since any search to detect materials in the parts-per-billion range also brings out other "interfering" substances.

Several years ago, two Swedish scientists at Stockholm University's Institution of Analytical Chemistry, Professor Gunnar Widmark and Soren Jensen, reported they had identified the other substances which were appearing during analysis of chlorinated pesticide residues. They said some of the materials were polychlorinated biphenyls or PCB. The amount reported was in the parts-per-billion range, or less. Since PCB's are not "broadcast" or spread around the land as are pesticides, the scientists theorized that the source must be the industrial wastes of PCB users.

Dr. Risebrough's more recent work reports the identification of PCB, along with DDT and DDE pesticides, in the tissues of birds and fish on the West Coast.

The conclusions of these scientists are puzzling from several aspects. Polychlorinated biphenyls are stable chemical compounds which are essentially insoluble in water. Their use does not make them easily released into the natural environment.

A principal market for PCB is in electrical applications where they are used as insulating fluids for transformers and capacitors. In this use, the chemical is completely sealed in metal containers. Another market is for heat transfer applications where the PCB fluid functions in a closed system.

PCB's are also used in several "plastic type" applications. Here the chemical is incorporated into the polymer as an integral part of the solid material. This applies whether the polymer is used as an adhesive, an elastomer or a surface coating.

The Swedish and American scientists also imply that polychlorinated biphenyls are "highly toxic" chemicals. This is simply not true. The toxicity of any material, whether it be chemicals, drugs, natural plants or even foods, is relative. Compared to the thousands of industrial chemicals and home products, PCB's are not toxic unless they are mishandled or misused. During more than thirty years of U.S. production and use, cases of any toxic effect have been extremely rare -- and then only where the simple precautions recommended for use were not followed.

To our knowledge, polychlorinated biphenyls are not sprayed or dusted on crops, woodlands or any other areas, as are pesticides. It is, therefore, not only puzzling, but extremely difficult to conceive how commercially produced PCB can show up in wildlife as DDT and other pesticides appear to be. This raises the question whether the substance identified in the Swedish work, and now in California, is actually PCB -- or whether they are materials which, due to the metabolism of other materials in the marine environment, appears to be PCB.

Unfortunately, even though techniques for analyzing tissue samples have become quite precise, the ability to analyze the possible impact of naturally occurring substances in the food cycle of living organisms has not made comparable advances.

Monsanto has a research program to confirm the identity of the compounds reported to be PCB by the Swedish and California scientists. We also cooperate, on a regular basis, with federal, state and university laboratories in their analysis of chlorinated hydrocarbon residues.

Additionally, Monsanto will continue to exercise the highest degree of control in its manufacturing, shipping and storage of PCB -- as we do with all products. In the functional fluids market, we have carried out a program for several years for the reclamation of used PCB's to avoid disposal of these valuable materials.

The source of the marine life residue identified as PCB is not yet known. It will take extensive research, on a worldwide basis, to confirm or deny the initial scientific conclusions. We will keep you advised of our own research and are available to answer questions as they may arise.

Very truly yours,

Elmer P. Wheeler
Manager Environmental Health

/ecd

Monsanto

ORGANIC CHEMICALS DIVISION

Monsanto Company
800 N. Lindbergh Boulevard
St. Louis, Missouri 63166
Phone: (314) 694-1000

February 18, 1970

DEPOSITION
EXHIBIT

#7 5157187
L.R.

Dear Sir:

Recently several newspaper and magazine articles have been published indicating that Polychlorinated Biphenyls (PCBs) have been discovered at some points in some marine, aquatic and wildlife environments. The quantities detected are said to be in the parts per million and parts per billion categories.

It is claimed that the PCBs found strongly resemble chlorinated biphenyls containing 54% and 60% chlorine by weight. Products which are sold by Monsanto under the trade names of Aroclor® 1254 and 1260 do contain chlorinated biphenyls. In addition to Aroclor® 1254 and 1260, Monsanto sells certain functional fluids containing Aroclor® 1254. These include Pydraul® 625, Pydraul® AC, Pydraul® AC - Winter Grade, Pydraul® 540, Therminol® FR-3 and certain dielectric formulations. Several other companies around the world also produce products containing chlorinated biphenyls.

As your supplier of Aroclor® 1254 and 1260 and formulated products containing 1254, we wish to alert you to the potential problem of environmental contamination as referred to in the newspaper and magazine articles.

We would like to point out the following additional facts:

1. Products such as Pydraul® 90, 135, 230, 312, A-200, F-9, 150, and 60, Turbinol® 153 and Therminol® FR-1 and FR-2 are not formulated with Aroclor® 1254 or 1260.
2. PCBs with a chlorine content of less than 54% have not been found in the environment and appear to present no potential problem to the environment.

47

1139

We have developed, and are now testing, new formulations to replace the Aroclor® 1254 and 1260 components in our Pydraul® products. The new products appear to be equal in performance and to have similar physical properties.

We feel that all possible care should be taken in the application, processing and effluent disposal of these products to prevent them becoming environmental contaminants. Of interest to you may be an article in Chemical Week, October 29, 1969, regarding water pollution standards set by each state in the Union. It is attached. This article reflects that good manufacturing practice in the future may require that no product used by any company should find their way into waterways.

We realize that you have marketed or may now market transformers and other electrical equipment containing dielectric fluids which include Aroclor® 1254 and 1260. Although these fluids are sealed into such equipment it is recognized that occasionally the fluid may be lost through leaks resulting from equipment misuse or equipment repair necessitating replacement of the fluid. Since the dielectric fluid contained in this equipment is only an incidental part of the over-all unit manufactured by you, we are not notifying the purchasers of such equipment of the potential environmental contamination problem described in this letter. We do recommend, however, that you notify such equipment users of this problem.

Sincerely yours,

Don Olson
Donald A. Olson
Director of Sales
Functional Fluids Group

1b

Attachment

1140

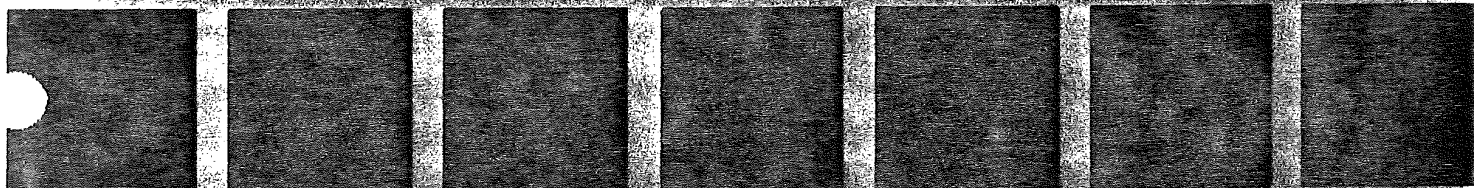
NIOSH

criteria for a recommended standard
occupational exposure to

DEPOSITION
EXHIBIT

#8 5157187
L.R.

POLYCHLORINATED BIPHENYLS (PCBs)



U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Public Health Service Center for Disease Control

National Institute for Occupational Safety and Health

WATER_PCB-SD0000059053

Trichloro
Corrected
copy of
attachment

Monsanto

MAR 24 1975

DEPOSITION
EXHIBIT

#9 5157187
L.R.

SPECIALTY & PROCESS CHEMICALS DIVISION

MONSANTO INDUSTRIAL CHEMICALS CO.
800 N. Lindbergh Boulevard
St. Louis, Missouri 63168
Phone: (314) 884-1000

March 18, 1975

Mr. Dan A. Albert
Staff Supervisor
Personnel Relations
Westinghouse Electric Corporation
Highway 58 West
South Boston, Virginia 24592

Dear Mr. Albert:

Attached are responses to the questions listed in your letter dated February 3, 1975.

In addition to the industrial hygiene practices described in our responses to your questions, I cannot overemphasize the need to properly control the use and handling of Inerteens to prevent their escape into the environment. Also, in discussing this information with your employees, I strongly recommend that the perspective gained from over 40 years of experience in which no human harm has resulted, be emphasized. In summary, the proper handling of Inerteens should pose no environmental or human health problems, permitting society's continued use of a very valuable material.

I hope the above information is useful to you. If I can be of further service please let me know.

Sincerely,

W. B. Papageorge

W. B. Papageorge
Manager, Product Acceptability
Specialty & Process Chemicals

WBP:pd

1626

1. Question: Does Inerteen have permanent effects on the human body? If so, what type of permanent damage and how long a period of time does it take for this to develop? If not, explain why, if possible.

The polychlorinated biphenyls in Inerteen can have permanent effects on the human body.

The length of time or period of exposure necessary to develop symptoms depends on the degree or amount of exposure. In general, a single exposure for a few minutes to atmospheric concentrations that cause irritation to the eyes and/or respiratory tract would not be expected to cause either the skin eruptions or demonstrable liver injury. The problem arises from repeated and prolonged exposure to atmospheric concentrations in excess of the accepted Threshold Limit Levels or repeated and prolonged skin contact.

The polychlorinated biphenyls have not been recognized as skin "irritants" in the same sense that caustic materials or many organic chemicals are irritants. Because of their "solvent" action in removing the natural fats and oils from the skin leading to drying and chapping, repeated and prolonged skin contact should be avoided.

When the polychlorinated biphenyls are mixed with other chlorinated hydrocarbons, the mixture may be classified as a skin irritant.

The potential toxic effects in humans from excessive exposure to polychlorinated biphenyls include injury to the liver and chloracne. In animals, the liver effect is demonstrated by increased liver weights and injury to cellular tissue. Although chloracne is difficult to evaluate in animals, in humans, this takes the form of comedones (large blackheads with typical acne pustules) and may be an external symptom of over exposure preceding serious liver injury.

Animal data and human experience indicate that the toxic effects are similar whether exposure results from ingestion, inhalation of vapors, or absorption of the liquid material through the unbroken skin.

(See AIHA Hygienic Guide Series - "Chlorodiphenyls" attached.)

2. Question: Several hourly employees have mentioned recently that many chemicals such as Inerteen cause sterilization after prolonged use. Is this true?

There is no evidence that polychlorinated biphenyls cause "sterilization" in humans.

3. Question: Since Inerteen affects birds and other animals, if there is no real effect to human beings, how do you explain it to employees in such a way that they will understand why it can kill a bird and not a human?

There is a potential real effect to humans - including death - as discussed in the answer to Question 1.

Due to differences in metabolism of food (and food contaminants) in birds and humans (and particularly the difference in the reproduction process in birds and mammals - including humans), birds are particularly sensitive to many chlorinated hydrocarbons including polychlorinated biphenyls.

4. Question: If an employee spills Inerteen on his clothing and later takes the clothing home to be washed with other clothes, will this have any effect on him or his family and should he carry his clothes home to be washed?

There should not be any effect on an employee or his family from home laundering of work clothing. If washed with other clothing, there may be residual odor of the chlorinated hydrocarbons in the clothing.

5. Question: Employees carry Inerteen home on the soles of their shoes and complain quite a bit about the effect Inerteen has on wearing out of their shoes. Is this a serious problem? Will Inerteen in the soles and leather of shoes, over a long period of time, have an effect on the feet and skin since the shoe is the only protective equipment we wear on our feet and the Inerteen penetrates through the leather.

There should not be polychlorinated biphenyl on the floor for workmen to contaminate their shoes to carry home. The plasticizer or solvent action will destroy or shorten the life of the shoes. More importantly, the wearing of contaminated shoes could lead to absorption of the liquid through the soles of the feet as through any other unbroken skin surface.

6. Question: There is one employee in our plant who had no problem whatsoever with Inerteen years ago. After six years of using, now when he works in Inerteen (which is a part of his job) he develops a swelling on the inner bicep of his left arm, only in one location. Could this be from Inerteen or not? It goes away as soon as he gets out of the Inerteen. It is similar to the swelling after taking an injection.

We do not believe there can be any association between "a swelling on the inner bicep" of the arm and exposure to polychlorinated biphenyls.

7. Question: Are there hand cleaning solvent materials that we should be using when working in Inerteen to coat our skin before working in it and to wash it off after we finish working in it? Please give your recommendation. Our employees working in Inerteen are not able to use gloves since it is an assembly area. Even if they could, the Inerteen would destroy the protective glove.

We assume the question refers to the use of "Barrier Creams" rather than a "hand cleaning solvent".

There are a number of barrier creams available to protect workers against water insoluble solvents. Probably the most effective include silicone "to provide an impenetrable shield". A problem with such creams is that they may offer a false sense of security.

Proper use includes a discipline which requires liberal application at the beginning of a work shift and after each washing of the hands during the work day.

WILLIAM B. PAPAGEORGE TESTIMONY
BAD AXE, MICHIGAN
MAY 1984

DEPOSITION
EXHIBIT
#10 5/27/87
L.R.

Δ - PAPAGEORGE

MR. CUBITT: I have no further questions.

THE COURT: Mr. Jungerheld?

MR. JUNGERHELD: Nothing further with Dr.

Willetts.

THE COURT: Mr. Davidson?

MR. DAVIDSON: Nothing further, Your Honor.

THE COURT: You may step down, sir.

Do you have Exhibit 421 and 422? These have been marked.
Go ahead.

MR. JUNGERHELD: Your Honor, we'd like to
call Mr. William Papageorge, please.

THE COURT: Very well.
Raise your right hand, sir.

W I L L I A M B. P A P A G E O R G E,
called at about 2:44 P.M. by the Defendant Monsanto,
sworn by the Bailiff, testified:

THE COURT: Take the stand, sir.

MR. JUNGERHELD: Your Honor, I don't
anticipate using any more overhead transparencies,
would you like me to remove those sheets?

THE COURT: If you don't mind.

DIRECT EXAMINATION

BY

MR. JUNGERHELD:

Q Would you give us your name, please, sir?

1 Q. And what's the date of those instructions?
2 THE COURT: Are you offering that exhibit?
3 MR. WOODWORTH: Yes, Your Honor.
4 THE COURT: What's the number on that?
5 MR. WOODWORTH: The number is 158. I'd move
6 for its admission.
7 THE COURT: Any objection, Mr. Jungerheld?
8 MR. JUNGERHELD: None, Your Honor.
9 THE COURT: Mr. Davidson?
10 MR. DAVIDSON: None, Your Honor. Thank you.
11 THE COURT: One fifty-eight is admitted without
12 objection.
13 MR. WOODWORTH: Thank you, Your Honor.
14 THE WITNESS: That date is July, 1964.
15 BY MR. WOODWORTH:
16 Q. And that has a stamp on it confidential. Is that to keep
17 the information within the company?
18 A. Yes.
19 Q. And would you read, please, then, what we have - first of
20 all on the very first page we have a title, Operating
21 Instructions as to Aroclor, is that right?
22 A. That is right.
23 Q. Okay. And then subsequent to that we get into an area
24 called Chlorine Gas, is that correct?
25 A. True. But it's under a broader heading.

Q. Okay. And go ahead and tell us what the broader heading...

A. The heading is - this is the safety and housekeeping section of the operating instructions.

Q. Okay.

A. And the first item referred to under that safety and housekeeping section is the Chlorine gas you refer to.

Q. And Mr. Papageorge, what was the chlorine gas used for?

A. It was used to provide the chlorine that is present in the finished product.

Q. That's what you bubbled through the benzene biphenyl or the biphenyl after it was made from benzene?

A. Correct.

Q. And so you had chlorine gas on hand on the premises for this process?

A. Yes.

Q. And how was it stored. Was it liquid, under pressure, or how? Obviously it was gas, because it says chlorine gas.

A. In 1964, the chlorine was generated at another unit as a gas and was transferred into the Aroclor unit and used directly.

Q. Chlorine gas itself is fairly toxic, is it not?

A. Oh, yes.

Q. And what, in fact, does it state for the very first causes and action to prevent as to chlorine gas? What's the one I have marked there with a little red pen?

1 A. It reads: Chlorine gas is a very toxic, corrosive gas.
2 Leaks are high pressure, can occur to liberate it.

3 Q. Okay. We're still on chlorine gas - does this next page
4 refer to chlorine gas yet?

5 A. No. The next page skips from number one item chlorine
6 gas, to number five item, fires.

7 Q. Okay.

8 A. It's another section of that safety and housekeeping
9 portion.

10 Q. And the next page is back on chlorine gas?

11 A. The next page appears to be a continuation of a reference
12 to chlorine gas. So I don't know what preceded that.

13 Q. Mr. Papageorge, I'm going to ask you to read - and this
14 is under safety and housekeeping as to chlorine gas and
15 as to biphenyls, the next topic under it. And if you'd
16 just read those three paragraphs, please.

17 A. Well, the first paragraph is under chlorine gas, and it
18 reads:

19 "If you are caught without a gas mask and large
20 amounts of chlorine gas are liberated, hold your
21 breath and go cross wind until you're out of range
22 of the gas. Once out of the area, obtain an
23 emergency gas mask and return to the area and
24 correct the problem."

25 Second paragraph:

1 "A victim of chlorine gas fumes must be removed
2 as quickly as possible from the gaseous area.
3 Place patient on his back with head and back
4 elevated. Milk may be used in mild cases as a
5 relief from throat irritation. If the patient
6 has stopped breathing, start artificial respiration
7 immediately."

8 Third paragraph:

9 Q And if I might interrupt for just a moment. The third
10 paragraph now refers to biphenyls, is that correct?

11 A Biphenyls.

12 Q Biphenyls?

13 A Correct.

14 Q And that's the product that was made by the uniting of
15 the benzene?

16 A That is correct.

17 Q And what's the safety and housekeeping recommendation or
18 instruction regarding biphenyl?

19 A It reads:

20 "Biphenyl is a flammable material which will burn.
21 Burning should not be done in the presence of
22 biphenyl or biphenyl vapors. The melting point
23 is 68.7 degrees centigrade, flash point 106 centigrade,
24 the fire point 124 centigrade. And the auto ignition
25 temperature is 258 degrees centigrade. Inhalation

1 of biphenyl fumes is not recommended since it
2 can cause a drug effect on the person."

3 Q. Thank you. And the next - as we go down this list, we come
4 to a subparagraph H referring to crude Aroclors.

5 A. Yes.

6 Q. Then if you would just read the last sentence under crude
7 Aroclors under paragraph 6H on page 6, please.

8 A. The last sentence reads:

9 "Prior to air blowing, the material has HCL fumes
10 entrained in it, which are irritating to the skin
11 or mucous membranes."

12 Q. Thank you. Now we finished that one for 1964, July 1964,
13 is that correct?

14 A. That's all I can see at the moment.

15 Q. And then we go on to another set, and this is WGK Operations
16 Manual. I imagine that's for the Krummrich plant?

17 A. That is correct.

18 Q. And it's kind of hard to read, but if you could bend around,
19 just the first paragraph of what's there of the first
20 paragraph, please.

21 A. Well, it's the - it seems like the last piece of a sentence
22 which I can only read, comma, but

23 "There is an explosion hazard if chlorine is
24 mixed with a strong-reducing agent, such as hydrogen
25 or hydrocarbons at high temperatures."

1 Q. Okay. And the next paragraph there, perhaps that's the
2 one I had marked.

3 A. "Under other hazards, this material causes
4 eye, skin, and mucous membrane irritation."

5 Q. Thank you.

6 A. It doesn't say what that material is, though.

7 Q. Okay. What's this - is that monochlorobiphenyl?

8 A. Yes.

9 Q. Okay. And that's one of the biphenyls, is it not?
10 It has one chlorine ring?

11 A. That is correct.

12 Q. What's the other hazards under that?

13 A. This material can cause dermatitis, systemic poisoning
14 from the fumes, and yellow atrophy of the liver.
15 There are skin, mucous membranes, and eye hazards
16 encountered in handling this material.

17 Q. Can you tell us what is meant, or what you understand is
18 meant by the term "yellow atrophy of the liver?"

19 A. I have an impression that it means the liver is damaged
20 and has an abnormal appearance as a result.

21 Q. And the paragraph under storage and handling for the
22 monochlorobiphenyl.

23 A. Storage and handling:
24 "Protect the skin, lungs and eyes by wearing
25 approved, protective clothing, respirator and

goggles. People who handle this material at elevated temperatures should have medical supervision and the air should be sampled to insure compliance with MAC."

Q. What's MAC?

A. I don't recall at the moment. I just don't remember.

"There are no special storage restrictions under normal conditions except store in a ventilated area."

Q. Thank you. Now we move to another set of operating instructions. And these are dated October, 1974, is that right?

A. That is right.

Q. And department 246 still operator, still operator would be the person who runs the distiller for Aroclors?

A. Yes.

Q. And department 246, where is that located?

A. This is at the WG Krummrich plant in Saugat, Illinois.

Q. Okay. If you'd read just the introductory paragraph on this under what's called Aroclors, please.

A. "Aroclors as manufactured in the Department 246 are non-flammable liquids which may vary greatly in viscosity and room temperature. Contact with skin should be avoided. In case of spills on the body, wash the effected area with soap and water."

Inhalation of Aroclor and Montar fumes should be avoided as they are highly toxic. Protect the skin, lungs and eyes by wearing protective clothing respirator and goggles. If spilled on clothing, change to clean clothes."

Q. Thank you. Next is another set of instructions, as to 246, again out of the Krummrich plant, is that correct?

A. It looks like 246, and an additional marking of some kind that's hard to read.

Q. Okay. And this is dated March, 1974, correct?

A. That is correct.

Q. And would you read that paragraph, the - what the operating instructions are for.

A. "These operating instructions are the property of Monsanto Industrial Chemicals Company and the recipient is responsible for its safe keeping. It contains confidential information of Monsanto Industrial Chemicals Company, which must not be reproduced, revealed to unauthorized persons, or sent outside the company without proper authorization. Either retain in the operating department or return to the standard documents department."

Q. Thank you. This is again Krummrich plant operating instructions department 246, and its still operator, is that right? The next group?

1 A. That is right.

2 Q I was going to ask you to read this paragraph, but it looks
3 like one that you've just read a short time ago, is it not,
4 as to Aroclors. In fact, the one you just read a short
5 time ago?

6 A. Yes. It does appear to be so.

7 Q This is again for the Krummrich plant operating instructions
8 department 246?

9 A. Yes.

10 Q And the date of this is June 1976.

11 A. Yes.

12 Q And the next set is from Krummrich, operating instructions
13 department 246, and what's this for?

14 A. It's an abbreviation, PREM.OP. That's premium operator,
15 that is the top worker assigned within that department.

16 Q And the next is standard manufacturing process, Aroclors,
17 Department 246, and that's dated 1972?

18 A. There's a written notation on it. It says 1972.

19 Q It also says before that, do not loan. Is that something
20 that typically would have been written on it?

21 A. I don't know that it's typical, but it's not surprising.

22 Q On the last page of this, Mr. Papageorge, under paragraph
23 G, there's Aroclor - a reference to that. And it says,
24 in parenthesis, see hygenic guide, series on chlorodiphenyls,
25 right?

1 A. Yes.

2 Q. Looking earlier in this particular process, it will state
3 something, for example, it says "chlorine, and it has
4 a description and formula, and contents, toxic hazard
5 rating, toxicology information.

6 A. Uh-huh.

7 Q. And it has the same on hydrochloride off-gas?

8 A. Yes.

9 Q. And it has the same thing previous on biphenyls. But when
10 we get to Aroclors, it says to refer to the hygienic guide
11 series. What's the hygienic guide series?

12 A. That was a publication that addressed the safety and
13 industrial hygiene of, in this case, chlorodiphenyls.

14 Q. And would that then be incorporated into the manual, so
15 to say?

16 A. Yes. A copy was attached and inserted in the manual.

17 Q. And who published that or where did it come from?

18 A. I don't recall the exact name of the group, but this was
19 a group of professional industrial hygienists that put
20 this information together for many products.

21 Q. The next set looks almost like a duplicate of the previous
22 set. Again, we have a standard manufacturing process,
23 Aroclors, department 246, and then in handwriting, do not
24 loan, 1972.

25 A. That's what it says.

1 Q And when we get to the last page, again for the reference
2 on Aroclor, it says see the hygienic guide series on
3 chloridiphenyls?
4 A Yes.
5 Q That would be the same one we just talked about?
6 A That is correct.
7 Q Now we have another one, another publication or a set
8 of instructions, process description for the manufacturer
9 of Aroclors in Department 246, is that correct?
10 A That is correct.
11 Q And I took that 66 there to mean 1966, would that be
12 reasonable? In fact, I'm sorry...
13 A It seems to be reasonable, yes.
14 Q It has a date written in, 1966, does it not?
15 A Yes. Yes.
16 Q And did you know these gentlemen here, by the way, that
17 signed this, that approved it?
18 A Yes.
19 Q Mr. Harris and the other gentlemen?
20 A Yes. Mr. Heisler, yes. Mr. Gibbs and Mr. Carder, yes.
21 Q And this has the same caveat, so to say, this process is
22 the property of Monsanto Company?
23 A That is a standard statement that appeared on all of
24 Monsanto's instructions.
25 Q Now this manufacturing process from 1966, if I can direct

1 your attention to Aroclor on page 39. On page 40, going
2 to Aroclor, and would you please read what the other
3 hazards are in relation to Aroclors?

4 A. It reads under other hazards:

5 "This material can cause dermatitis, systemic
6 poisoning from the fumes and yellow atrophy of the
7 liver. There are skin, mucous membranes and eye
8 hazards encountered in handling this material."

9 Q. And what is systemic poisoning?

10 A. I have an understanding. I don't know if it's accurate.
11 But this is a reaction by the body to a foreign material,
12 and it's in the system - in the body's system.

13 Q. It's a poison to the body, in other words.

14 A. That's one way to look at it, yes.

15 Q. And this looks like a repeat of the previous one?

16 A. Yes.

17 Q. Back here, Mr. Papageorge, I put a question mark on that
18 because it says old SMP Aroclors?

19 A. It does say that, yes.

20 Q. What would SMP mean?

21 A. Standard manufacturing process.

22 Q. Okay. We have a - there's a first sentence, I think, to
23 that other paragraph that you couldn't read before.

24 A. I see.

25 Q. Would you read that, please? Just that first paragraph?

1 A. It says:

2 "Diphenyl and Aroclor are toxic materials. To
3 prevent toxicity...

4 End of statement.

5 Q. Okay. And then go to the next page.

6 A. "Clean change of clothes is furnished daily
7 to each employee and time is allotted at the
8 end of each shift for bathing. In addition, one
9 should wash his hands and face before eating."

10 But I don't know if the two are necessarily related.

11 Q. Okay. Would the reason for recommending to the employees
12 to wash their hands and face before eating is so they
13 wouldn't ingest this material?

14 A. Yes. That a - this is a fairly common statement for a
15 chemical manufacturer.

16 Q. To protect the health and safety of your workers?

17 A. That is right.

18 Q. Would you read this paragraph, please, under the hazards
19 and means to safeguard material?

20 A. "There are many accidents that can occur that
21 may be classed as safety and health hazards. These
22 accidents may result from such things as broken
23 process lines, gas leaks, acid leaks, steam burns,
24 etcetera. The following safety equipment is provided
25 to each employee: One pair of rubber overall, goggles,

one pair spectacles, one pair rubber shoes, one pair rubber-covered gloves, one pair of canvas gloves, and one fume respirator. And for the department use, one gas mask, one clean change of clothes, four safety showers, and one stretcher."

Q. Okay. Thank you. And this, again, appears to be a duplicate of the prior one, OMP Aroclors?

A. It looks the same, yes.

Q. And we have that same sentence beginning?

A. Right.

Q. And would you read just from the very last page, then, the hazards paragraph? Just the first paragraph of the hazards?

A. "The fumes from Aroclor 1254 are quite obnoxious and are, when inhaled excessively, dangerous and hazardous. When handling this material in relatively closed areas, ample ventilation (hoods in extreme cases) should be provided for removal of the fumes from the working area. If fumes attack the mucous membrane of the nasal passages and irritation of the membranes and slight lung congestion may result from excessive inhalation of the fumes."

Q. Thank you.

THE COURT: In light of the hour, members of the

DEPOSITION
EXHIBIT

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L.C.

April 8, 1970

Dr. Herbert Blumenthal
Chief, Petitions Review Branch
Bureau of Foods, Pesticides and
Product Safety
U. S. Food and Drug Administration
200 C Street, Southwest
Washington, D. C. 20204

Dear Herb:

I thought you would be interested in knowing that Monsanto is committed to a program which will allow the future use of our Aroclors only in those applications where escape to the environment can be prevented.

Secondly, we have essentially a crash program under way to find non-persistent, non-chlorinated substitute products. Already, we have two large users switching to substitutes of this type. Obviously, Herb, you can recognize the competitive aspects of developing and phasing into sales such type of substitutes so, at present, we are not making any widespread announcement. On the other hand, if the publicity gets as red hot as it did in the case of DDT, we may be forced to make such an announcement at any time.

Cordially,

R. Emmet Kelly, M. D.
Medical Director

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