

1 IN THE CIRCUIT COURT
2 TWENTIETH JUDICIAL CIRCUIT OF ILLINOIS
 ST. CLAIR COUNTY

3 FRANCES E. KEMNER, et al)
)
4 Plaintiffs,)
)
5 VS.) No. 80-L-970
)
6 MONSANTO COMPANY,)
)
7 Defendant.)

8
9 Before the HON. RICHARD P. GOLDENHERSH, Judge

10
11 REPORT OF PROCEEDINGS

12 JURY TRIAL

13 November 6, 1985

14
15 APPEARANCES:

16 MR. REX CARR and MR. JEROME SEIGFREID
 on behalf of the Plaintiffs;

17 MR. KENNETH R. HEINEMAN and MR. JOSEPH NASSIF
 on behalf of the Defendant.

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19
20 KIMBERLY GANZ, CSR, RPR, CM
 Official Court Reporter

1 BE IT REMEMBERED, that on November 6, 1985 the same
2 being one of the regular judicial days of said court, the
3 above-entitled cause came on regularly for hearing before the
4 HONORABLE RICHARD P. GOLDENHERSH, one of the Judges of said
5 court, at the St. Clair County Building, 10 Public Square, in
6 the City of Belleville, St. Clair County, Illinois.
7 Whereupon the following proceedings were had:

8 (The following proceedings were had in the hearing
9 and presence of the jury).

10 THE COURT: I apologize for starting late. There
11 were some other court matters to be taken care of.

12 FRANK DOST
13 having resumed the witness stand, being previously sworn,
14 testified further as follows:

15 DIRECT EXAMINATION

16 By

17 MR. KENNETH R. HEINEMAN.

18 Q. Doctor Dost, when we stopped off, stopped testimony
19 yesterday afternoon, you were just, you had just finished
20 discussing these photographs of Doctor Zahalski's charts
21 which are marked as Defendant's Exhibits 1291 through 1295.
22 In the meantime, have you done some writing on these
23 photographs?

24 A. Yes. I made some remarks that relate -- I wrote

1 some remarks on there that related to the oral testimony that
2 I gave yesterday with respect to those diagrams.

3 Q. Would you step down, please, and with respect to
4 each of the exhibits, point out to the jury the remarks that
5 you have written on them?

6 A. The diagram as it is set up in my view at least
7 does not represent the process by which TCDD is associated
8 with an increased incidence of tumors in experimental
9 animals. There is some resemblance to the process by which
10 TCDD interacts itself to induce the P450 enzymes that react
11 with foreign chemicals. The interaction to this point is
12 correct except that I do not believe that the term recognizer
13 is appropriate because it is not specific. It is a receptor
14 that accepts a large number of chemicals and that complex is
15 formed in the body of the cell, not on the cell wall.

16 Q. Which number is that that you were just referring
17 to?

18 A. This was on Exhibit 1291, the first of that
19 series. In 1292, you really don't have such specific
20 information that we could say it reacts with a critical
21 target. Obviously it has to react in a special way in order
22 to cause the sequence of events that take place afterwards
23 but that interaction or the nature of that interaction at the
24 present time is not adequately known. There is information

1 about it but no one has taken the position that they
2 understand precisely what is happening.

3 In any case, there is an interaction with the
4 genome to cause this RNA blueprint that we have talked about
5 to go to the endoplasmic reticulum. These are the
6 messengers, so called. That is not an inappropriate way to
7 describe messenger RNA because that is what the biochemists
8 call it because the purpose is to carry information from one
9 place to another. Out in the cell, there is interaction on
10 the messenger RNA causes the endoplasmic reticulum to
11 manufacture specific proteins. These proteins are not, this
12 represents the same thing as the receptor or recognizer that
13 was described earlier. And that is stated here.

14 The proteins that are made are not receptors. The
15 proteins are made are the P450 enzymes and other enzymes that
16 are not in that particular chemical class that are formed
17 that we have, many of which we have named and they remain on
18 the endoplasmic reticulum which is a very, a membrane that is
19 all wrinkled up in the cell. Very high surface area that
20 occupies a relatively small part of the space in the cell.
21 If you were to take a big piece of tissue paper, you could
22 crinkle that all up into a very, very small space and yet the
23 surface area is still very, very large. The surface is still
24 the same. It just occupies a very small space and that is

1 the way many of the membranes inside the cell are formed so
2 that they can get an awful lot of surface into a very small
3 space so that endoplasmic reticulum is in the cell sap or in
4 the soluble or out in the cell body and not out here on the
5 cell wall. And we are not talking about a receptor.

6 These are the proteins that are made are enzymes
7 that among other things have the function of interacting with
8 TCDD and making it less toxic. We don't go back to this
9 cycle a second time. And we don't create additional -- as I
10 said, you don't create additional receptors but then
11 apparently according to this description, do something
12 different than they did the first time. And we don't form
13 reactive intermediates. This statement is incorrect.

14 And then in the last diagram, again the question of
15 reactive intermediates and they don't bind in the cell. The
16 results of the interaction of TCDD with those enzymes is a
17 non reactive product and as I stated the other day, I don't
18 really think that any mechanism of this sort represents any
19 of the, any of the mechanism by which TCDD is associated with
20 the increased tumor incidence that is seen in high lifetime
21 doses in the various studies that have been done to show that
22 TCDD has some potential presumably as a promoter.

23 Q. Doctor Dost, then the last one down by the word
24 transformation, you put a question mark there, did you not,

1 sir?

2 A. Yes.

3 Q. And what was the purpose of that?

4 A. Well, for one thing, we believe that TCDD is a
5 promoter. There are some problems with terminology because
6 the term, the concept of promotion and transformation
7 sometimes become -- I won't say they become interchanged but
8 it is hard to tell where one leaves off and the other begins.
9 We do not know anything about a mechanism by which that might
10 happen and it certainly doesn't occur as a function of
11 binding of TCDD to critical targets, so to speak, such as
12 DNA. Certainly we know that the formation of the metabolites
13 of TCDD does not result in cell toxicity. Apparently it is
14 toxicity that is produced by TCDD depends on in tact,
15 unchanged TCDD molecules.

16 MR. HEINEMAN: Your Honor, at this time we would
17 move the admission of Defendant's Exhibits 1291 and 1295 into
18 evidence.

19 MR. CARR: No objection, Your Honor.

20 THE COURT: 1291 and 1295?

21 MR. HEINEMAN: 1291 through 1295.

22 THE COURT: All right. Admitted without objection.

23 Q. Doctor Dost, in describing or in discussing these
24 particular charts, you will recall that Doctor Zahalski

1 talked about one molecule of dioxin being able to initiate
2 cancer or to initiate immune collapse. Do you recall that,
3 sir?

4 A. Yes.

5 Q. Now, did you tell us yesterday you disagreed with
6 that?

7 A. I did, yes.

8 Q. And why is it that you disagree with that, Doctor
9 Dost?

10 A. Well, we can deal with it biologically or
11 chemically or both. To begin with, reactions of chemicals in
12 the body follow the same rules, the same natural laws that
13 reactions in a test tube or anywhere else must follow. And
14 one fundamental concept, one fundamental fact in chemical
15 reactions is that as the concentration of reactants
16 decreases, the rate of reaction decreases. In fact, it
17 decreases more rapidly than the concentration does and also,
18 the probability that a reaction can be started in the first
19 place also depends on concentration. I believe I pointed out
20 yesterday that one of the reasons for this is because it
21 takes time. It is very difficult to imagine time in
22 compartments so small that it would relate to a molecule. I
23 mean we talked about how many molecules there are in very,
24 very small amounts of any substance yesterday. However, if a

1 molecule does not remain on the site where it is going to
2 react for some finite period of time, and the physical
3 chemists speak in terms of picoseconds when they discuss
4 these kind of things. If it doesn't stay there long enough,
5 it won't react.

6 Well, it can stay there long enough if there are
7 enough other similar molecules in the system to make it stay
8 there. Otherwise, because of the natural physical activity,
9 molecules move around. They don't stay in one place. They
10 are not lying there like a rock. And as a consequence, the
11 molecule will not stay in place long enough for the physical
12 forces that would hold it in place to actually bind it so
13 that it can react. If there is a large population of
14 molecules that surround it, then the potential for it to stay
15 in place long enough to react becomes significant and a
16 reaction presumably can take place. It may be very slow but
17 it can take place. A single molecule, the likelihood of a
18 single molecule reacting with anything is in practical terms
19 impossible.

20 Q. What about with respect to promotion of an already
21 initiated cancer cell?

22 A. Okay. We are talking about, again we are talking
23 about a chemical reaction and TCDD is not a magic chemical.
24 There are no magic chemicals. There are no chemicals that

1 behave apart from the basic laws of chemistry and physics and
2 the same idea applies. There just is not enough material
3 there even with the incredible toxicity, if you will, of TCDD
4 and it is a very toxic substance. No one has ever denied
5 that. There has to be enough molecules available to
6 interact. One molecule in a practical reasonable sense is
7 not going to undertake a reaction.

8 Q. What about individual susceptibility? You would
9 certainly agree that one individual may be more susceptible
10 to the action of the chemical than another person may be?

11 A. The differences in sensitivity of individuals are
12 really not all that great. You may be talking about
13 differences of 10, 20 fold, even animals or people with
14 genetic differences that magnify individual or variability
15 within a species. These differences are not on the order of
16 hundreds and thousands and ten thousands. They are
17 relatively small numbers. Now to be sure, an individual who
18 is twice as sensitive, let's say, to an anesthetic agent and
19 this is a very real possibility. A person who is twice as
20 sensitive to an anesthetic agent is always a possibility in a
21 surgical context and the anesthesiologist must be exceedingly
22 careful that that individual is not just given some dose of
23 anesthesia and assume that they are going to be properly
24 anesthetized because the average dose is at some level. If

1 an individual is only twice as sensitive, the consequences
2 could be drastic but a 10 fold sensitivity, when we are
3 speaking in an issue where levels are so low as to be
4 virtually unimaginable has very little meaning.

5 Q. So that you wouldn't find a ten trillion fold --

6 A. Oh, no.

7 Q. Difference in sensitivity?

8 A. No. Never.

9 Q. Now, with respect to the idea that we discussed
10 yesterday that Doctor Zahalski suggested that promotion would
11 occur before initiation. Do you remember that testimony?

12 A. Yes.

13 Q. Are there any studies, particularly in skin tumor
14 agenesis in animals that have looked at what happens when
15 TCDD is applied to the skin before a known initiating
16 carcinogen is applied?

17 A. Well, the skin tumor model is a very widely used
18 system for studying the ideas, the processes of promotion and
19 initiation and their interrelationship because -- and the way
20 that the system works is that an initiator, let's say
21 di-methylbenzanthracene. It is applied to the skin and then
22 the promoter is applied to the same spot over an extended
23 period. Let's say twice a week for half a year after a
24 single dose of an initiator. And this is a very sensitive

1 system for at least certain kinds of tumor agenesis and it
2 has provided a great deal of information that has given rise
3 to the concepts of promotion and initiation.

4 TCDD doesn't work as a promoter in that rather
5 sensitive system. In other words, if you were to treat a
6 mouse with di-methylbenzanthracene and then follow it with
7 TCDD at, well, the doses that have been used are as high as a
8 microgram twice a week applied to the spot over, say, a 30
9 week period, still don't see any promotion. You see some
10 skin injury. Some hyperplasia but you don't see any tumors.
11 If you give TCDD prior to the initiator in that system, what
12 happens is very dramatic. The incidence of tumor formation
13 drops very sharply and the reason that it drops is very
14 straight forward. What TCDD has done is to initiate the
15 chemical metabolizing enzymes, the P450 enzymes in skin where
16 they exist, not as extensively in liver but they are there
17 and so if we give TCDD, let's say a week ahead of the
18 initiator, the skin, the P450 enzymes in the skin have risen
19 sharply. They have been induced and they metabolize the
20 di-methylbenzanthracene and get rid of those of it and so the
21 tumor incidence drops very dramatically.

22 Q. So instead of promoting, they inhibit?

23 A. In that situation instead of promoting, they really
24 inhibit because they are accelerating the metabolism of the

1 initiating carcinogen so that the actual availability of the
2 initiator has been decreased very sharply.

3 Q. Now --

4 MR. CARR: Counsel, did the witness perform these
5 studies? You said they were studies but I didn't hear him
6 identify --

7 A. I did not do that.

8 MR. CARR: I didn't hear you identify who did it?

9 A. Those have been done by Doctor Berry, Doctor Slaga
10 again. A group primarily at the National Institute of
11 Environmental Health Sciences. DeGiovanni, Cohen.

12 Q. Now, discussing di-methylbenzanthracene, I will
13 have you take a brief look once again at the Eisen chart
14 which is Plaintiffs' Exhibit 233. This particular document,
15 this particular study, is this TCDD specific? Is that
16 what --

17 A. This study is looking at -- No, it is not. Many of
18 the things that are in here do relate to TCDD. I believe I
19 mentioned yesterday that this part clearly is associated with
20 the action of TCDD and this part. There are questions as the
21 document shows but whether these particular enzymes are also
22 elevated, the evidence is not clear. It doesn't apply to
23 this concept of the formation of a reactive intermediates
24 which then interacts to cause cancer or other elements of

1 toxicity. There is a possibility that there is evidence, in
2 other words, to suggest that TCDD does have some activity in
3 this general area. This is an area that has not been fully
4 worked out yet. I suspect that we will find, in fact, there
5 are interactions here and establish those very clearly.

6 Q. The question mark, sir, do they mean that it is a
7 hypothesis?

8 A. Well, as I said yesterday, this is an overall model
9 that is intended to include all, most of the reasonable
10 possibilities for all chemicals but not as a specific model
11 for any one chemical. You mentioned di-methylbenzanthracene.
12 That fits into this category because there are presumably
13 reactive intermediates formed. However, in the studies that
14 I just was describing, apparently the interaction in the skin
15 is sufficient that it either, there is such a short-lived
16 intermediate or conceivably there may be a direct conversion
17 without going through the reactive intermediate. That
18 substance does cause or does form reactive intermediates in
19 the liver.

20 Q. Now, there have been a number of occasions here,
21 Doctor Dost, when you have disagreed with the testimony of
22 Doctor Silbergeld and with Doctor Zahalski. The views you
23 have expressed in disagreement with those two witnesses, are
24 those views solely your own, sir?

1 A. No. They are not.

2 MR. CARR: Your Honor, I object, certainly object to
3 that unless the witness is going to be here to cross
4 examine. I object as to what Doctor Dost is doing and what
5 Mr. Heineman is doing and counsel clearly knows that this is
6 not proper.

7 THE COURT: Objection is sustained. It is not
8 proper.

9 Q. Your views here today, sir, and yesterday, what
10 have you based your opinions on?

11 A. My opinions are based on a thorough analysis of the
12 scientific literature, analysis of the opinions of other
13 scientists.

14 MR. CARR: Your Honor, I most certainly disagree,
15 object to that. Counsel knows that it is not proper. If
16 there is an authoritative article or if there is a citation
17 of some sort, this witness may refer to it. He may not refer
18 to conversations of so-called other scientists that are not
19 here. We have no way of identifying. No way whether it is a
20 fact or truth. It is the rankest form of hearsay and counsel
21 knows it from many times we have been up on points similar to
22 that and I object to it and ask the jury to be instructed to
23 disregard what this witness has said with regard to
24 conversations with other scientists.

1 MR. HEINEMAN: May I respond to that? As we have
2 gone through this witness's testimony, he has specifically
3 related to specific papers and specific studies by specific
4 scientists when he has expressed his opinion.

5 MR. CARR: And I have no objection to that. That is
6 not what I am objecting to now.

7 THE COURT: That is not a similar situation. The
8 objection is sustained. The jury is ordered to disregard the
9 remark. Mr. Heineman, you were admonished to avoid any such
10 area, any such remarks or any question that calls for such
11 remarks.

12 Q. Doctor Dost, have you, yourself, done any specific
13 hands-on work in the laboratory with dioxin?

14 A. No. I have not.

15 Q. But you said to us that you have read the
16 literature?

17 A. That is correct.

18 Q. On the subject of dioxin?

19 A. Yes.

20 Q. How many -- approximately how many such articles
21 have you read?

22 A. I have no idea, really. Hundreds, I am sure.

23 Q. Is it -- do toxicologists such as yourself in
24 operating daily as toxicologists deal solely with the result

1 of their own work in reaching conclusions about situations
2 that are presented to them?

3 A. No. That would be impossible because for a number
4 of reasons. For one thing, toxicologists tend to do research
5 on certain classes of chemicals or work involving certain
6 kinds of mechanisms. It is not possible for any of us to do
7 a significant amount of work in any specific field. I at one
8 time was considered to be a major expert in the area of the
9 toxicology of the hydrosenes and I doubt if I had done three
10 percent of the research on hydrosenes. The toxicologist has
11 to be in a position to respond to problems and use the basic
12 principles, the basic scientific principles and concepts of
13 toxicology in doing that and there is no way that any of us
14 could possibly do all of the work or even enough work to
15 reach a conclusion on a wide basis. Certainly a researcher,
16 Allen Poland, for example, has done a great deal of work on
17 the AHH receptor and I am sure he has a very clear conclusion
18 in his mind based on his own work about that but I am also
19 sure that it, he has examined everybody else's work as he has
20 approached his conclusions and we are all -- When we do do
21 research, our research is shaped by the work that other
22 people have done. If it was not, we would just waste our
23 efforts because everything would be done over and over again.

24 Q. Now, one of the things I would like to ask you

1 about, sir, is the testimony by Doctor Zahalski on May 3rd of
2 1984, page 110. He suggested that one molecule of dioxin
3 could result in one million cancer cells in 30 days. Do you
4 remember that testimony, sir?

5 A. I remember that testimony.

6 Q. Do you agree with that?

7 A. No.

8 Q. And could you tell us why not?

9 A. Well, I have already explained why I really am
10 convinced that a single molecule is not going to induce the
11 carcinogenic process. But some simple arithmetic would and I
12 discussed yesterday also the doubling time of tumor growth.

13 If I recall, Doctor Zahalski had mentioned the idea
14 of rewarding his daughter for something, a penny a day,
15 double it tomorrow, or do you want that or do you want some
16 now. If one were to use that figure, that is using a
17 doubling time of one day where there are two cells, one cell
18 yesterday and two day, four tomorrow, et cetera, there would
19 be on the order, I have calculated this, it would be in
20 excess of 500 million cells at the end of a month. If we
21 assume a doubling time or if we assume that there are a
22 million cells starting from that single cell, at the end of a
23 month, there would be, if we did start just with one cell, at
24 the end of a month there would be a million cells, at the end

1 of two months there would be a trillion and at the end of
2 three months there would be, each of those cells would form a
3 million cells and there would be 10^{18} cells. Now, if
4 we assume that there are 10^9 , that is one billion
5 cells in a gram of tissue which is something of the order
6 that we find in the liver, that means that at the end of the
7 second month, there would be a thousand grams of tumor. Now,
8 a thousand grams is only a kilogram. That would certainly be
9 noticeable, but by the end of the third month, we would have
10 a thousand kilograms of tumor cells and a kilogram is a
11 fairly substantial amount of material. A person may weigh 70
12 or 80 kilograms and by the third month we already have a
13 thousand kilograms of cells. If we wanted to multiply this
14 by the amount of, by the number of molecules that we were
15 talking about yesterday that are in the body at a dose of one
16 nanogram per kilogram and I don't remember what the number
17 was but we were talking something on the order of 10^{13}
18 trillion molecules at that dose, I think, something of that
19 sort and if each of those molecules was to initiate this
20 process, we are moving into numbers here that I no longer am
21 able to imagine.

22 But in three months, a single cell reproducing at
23 that rate would produce a tumor that weighs about a thousand
24 kilograms.

1 Q. How much is that?

2 A. Well, that is 2200 pounds, something over a ton.

3 Q. Now, and that would be if indeed one molecule will
4 result in a million cancer cells in 30 days?

5 A. Yes.

6 Q. Now, in fact, sir, would you explain to the jury
7 the doubling rate that occurs in cancer and how that relates
8 to a latency period and the development of a tumor after a
9 period of time?

10 A. Well, the latency -- latency is defined in
11 sometimes different ways because you recall, if I might bring
12 up 1285, we talked about this initiation process and that can
13 happen and that cell may remain there initiated for the life
14 of the animal even. So there is no specific time between
15 this event and the process of promotion and ultimately
16 transformation. That cell may remain initiated for a
17 lifetime. That sometimes is thought of as part of the
18 latency period but if we just think of it from this point
19 forward when that cell has reached the point where it is
20 going to become a tumor without question. It has evaded
21 immune surveillance, it is going to become a tumor and if we
22 have, if we have a doubling time, I think I spoke yesterday
23 of doubling times on the order of 30 to 90 days.

24 Now there are some culminating types of tumors that

1 go at an incredible rate. Not doubling everyday but they do
2 go very fast but if a tumor doubled at a rate of 90 days, at
3 the end of one year we would have two, four, at the end of
4 six months we would have four cells. At the end of nine
5 months we would have eight and at the end of 12 months we
6 would have 16 and so on. And it would probably require a
7 population. If one was really looking and was able to see
8 tumors, you have got, you know, tumors are hardly ever found
9 until they are of substantial size because they are found
10 only when they are starting to cause some kind of
11 disruption. A tumor of this size in the liver would almost
12 certainly be undetected but with our present level of
13 sophistication because it is hard to identify tumors on the
14 basis of some biochemical change in the body. The only way
15 they can really be identified is morphologically in most
16 cases.

17 Now, some of the leukemias and so forth are
18 detectable with a blood sample, but even so, they are rarely
19 sought. You rarely look for them unless something is going
20 wrong. So, most of the time a tumor isn't detected until it
21 is causing some kind of a disturbance and that may be because
22 of its size and it takes a long time, if a tumor is, let's
23 say, this big around, it probably weighs what 20 or 30 grams
24 and if there are a billion cells per gram, that means 20 or

1 30 billion cells and when the rate of doubling is only 90
2 days, that means that the growth of that tumor is going to be
3 very, very small and it will probably have been going on for
4 years and years and years. There are tumors that double even
5 more slowly than that. So, that really is the basis for the
6 latency idea. It is a little depressing to realize that all
7 of this can be going on without us knowing it but that is
8 what happens.

9 Q. Now, at some point, though, the mass of cells gets
10 large enough that a doubling has a very substantial effect,
11 isn't that right?

12 A. Oh, yes. There is another property here, too, and
13 that is as a tumor becomes very large, oftentimes the
14 doubling rate slows because the tumor is not well nourished.
15 In other cases, it is well nourished and it will just keep
16 right on going but oftentimes the tumor may be even
17 capsulated. The cells in the tumor are not necessarily well
18 nourished and that would slow the growth down. It varies
19 with the kind of tumor.

20 Q. Now, with respect to the dose response theory, what
21 do the studies of the effect of TCDD and cancer, what do they
22 show with respect to whether or not a dose response principle
23 exists in cancer?

24 A. Well, they all clearly show that at the highest

1 doses, there is some incidence of increased, some increased
2 incidence of tumors, particularly in the liver, some
3 thyroid. And in animals that are fed the material,
4 apparently some incidence of tumors in the palate and upper
5 respiratory area but as the dose decreases, and I might add
6 that at those doses, these are animals that are showing other
7 kinds of pathology as well. And as the dose decreases, that
8 evidence just simply stops and at the lower doses, there is
9 no evidence of increased tumor incidence.

10 Q. Well, who are the people? Who are the scientists
11 who have studied this subject?

12 A. There have been a series of studies done under the
13 auspices of the National Toxicology Program. They have
14 studied TCDD in rats and mice given by stomach tube and also
15 applied to the skin. The study by Koceba in rats that I have
16 mentioned a number of times. Studies by a researcher named
17 Toth, T-o-t-h, on mice. There have also been studies -- I
18 mentioned earlier the skin tumor initiation promotion
19 experiments that have been done. Allen Poland has done
20 studies in nude mice. Identifying the promotion character of
21 the promoting character of TCDD as have Henry Pitot. Those I
22 think are the principal sources of data. There has been a
23 study done by at the University of Wisconsin by Van Miller,
24 James Allen and that group but it was a peculiar study, very

1 small numbers of animals. It was intended as a preliminary
2 study. They found evidence of tumors but most of the control
3 animals died. There was a higher mortality in the control
4 animals than there was in any of the experimental groups
5 except those in which all of the animals died because of the
6 high dose of TCDD.

7 They used a very wide range. I think they used
8 nine different dose levels but a relatively small number of
9 animals in each.

10 Q. When they are measuring lethality of animals, do
11 they find that it is cancer that kills them?

12 A. It doesn't seem to be, no. That is not the
13 mechanism by which they die.

14 Q. With respect to whether there is a threshold for
15 TCDD and cancer, have the Koceba studies addressed that
16 subject?

17 A. Koceba did not specifically address that as part of
18 the problem. The data suggests that there is, in fact, a
19 threshold. In my opinion, I think there is a threshold for
20 that kind of effect.

21 Q. What doses did Doctor Koceba test and what did he
22 find with respect to any kind of tumors?

23 A. Well, in his two year study, they used a dose or a
24 dose rate, if you will, of a microgram per kilogram per day.

1 I beg your pardon, a tenth of a microgram per kilogram per
2 day over the lifetime of the animal and then there was a dose
3 ten fold lower than that at .01 micrograms and a dose at .001
4 micrograms per kilogram per day.

5 Q. And what was found at those respective doses?

6 A. At the lowest dose, no evidence of any carcinogenic
7 response. At the intermediate dose, there were some hepatic
8 nodules that are characteristic of a lot of hepatic
9 intoxicants and then at the higher dose, primarily hepatic
10 carcinomas, some evidence of tumors, increased tumor
11 incidence as I have just mentioned in the palate and upper
12 respiratory tract. I don't recall whether he found -- I
13 think he may have found some evidence of thyroid tumors in
14 the high doses too and decreased incidence of tumors in the
15 endocrine gland.

16 Q. Lower than in the control?

17 A. Yes.

18 Q. Now, based upon that, do you have an opinion as to
19 whether there is a no effect level with respect to TCDD and
20 any relationship to cancer?

21 A. Well, in my opinion, I think that there is a no
22 effect level.

23 Q. And what is that?

24 A. Well, based on the studies of Koceba and in the

1 National Toxicology Program studies where the doses were
2 approximately equivalent and in the Toth studies on mice, I
3 think that we can consider .001 micrograms per kilogram to be
4 a no effect level. It is often stated as a no effect level.

5 Q. Now, Doctor Dost, there has been some testimony in
6 this case in the past relating to a certain paper put out by
7 the Centers for Disease Control and Doctor Renate Kimbrough,
8 Henry Falk and others. And, in fact, it is in evidence as
9 Plaintiffs' Exhibit Number 1255. Have you read that
10 document?

11 A. I have, yes.

12 Q. And what is the level in soil that is discussed as
13 a level of concern in that document?

14 A. In that document, they consider that a level of one
15 part per billion or above in residential soil is a level of
16 concern.

17 Q. I would like you to look at the exhibit -- first of
18 all, I have a copy of Plaintiffs' Exhibit 1255 and I would
19 like to direct your attention to page 74 of that exhibit.
20 Doctor Dost, I am showing you Defendant's Exhibit Number 1296
21 and ask if that is an accurate copy of page 74 of Plaintiffs'
22 Exhibit 1255?

23 A. Yes. It is apparently a photocopy of this page.

24 Q. All right.

1 MR. HEINEMAN: Your Honor, we would move admission
2 of Defendant's Exhibit 1296.

3 MR. CARR: No objection.

4 THE COURT: Okay. It is admitted without
5 objection. Before you get into that, let's take a short
6 break at this point in time. Court is in recess.

7 COURT RECESSED:

8 (The following proceedings were had in the hearing
9 and presence of the jury)

10 FRANK DOST

11 having resumed the witness stand, being previously sworn,
12 testified further as follows:

13 DIRECT EXAMINATION

14 By

15 MR. KENNETH R. HEINEMAN.

16 Q. Doctor Dost, you have before you there Plaintiffs'
17 Exhibit 1255, do you not, sir?

18 A. Yes, I do.

19 Q. And the one part per billion level in residential
20 soil that you related to us, what according to the CDC is the
21 relationship between the level of contamination and where
22 that contamination is in terms of the assumptions they have
23 made and the level that they have made applicable?

24 A. The one part per billion level relates to

1 residential soil. The assumption is that the entire area is
2 contaminated at a level of one part per billion. In other
3 words, this is an area where people are living and the entire
4 area is assumed to be contaminated at one part per billion.

5 Q. Now, by the entire area, you are referring, the
6 articles is referring to what?

7 A. It is referring to soil. In other words, the
8 garden, the lawn, wherever soil contact might be and the
9 assumption is that it could very well be the entire area in
10 open soil.

11 Q. Does it state anything with respect to whether any
12 commercial or industrial area is considered?

13 A. Yes, it does. It points out that in commercial or
14 industrial areas where there is often gravel covering,
15 paving, where there is essentially limited foot traffic
16 contact by individuals, people may be crossing over it but
17 they are not in contact with the soil to any appreciable
18 extent, they state that higher levels would be also without
19 risk. In other words, they would not, they would be
20 concerned only at higher levels than one part per billion.

21 Q. Did they state whether or not inhalation or skin
22 contact would be assumed in gravel or commercial sites?

23 A. They did speak to that and their assumption was
24 that there would not be skin contact nor certainly there

1 would not be ingestion because the people who are going to be
2 in industrial sites are going to be almost exclusively adults
3 and small children are not going to be playing in such sites.

4 Q. Does this one part per billion, sir, relate to a
5 concentration in a product or a concentration in soil?

6 A. No, sir. It relates only to the ultimate
7 concentration that might result in soil.

8 Q. And what were the assumptions behind the one part
9 per billion?

10 A. Well, they have listed them on page 74, yes. Which
11 we have reproduced here. The assumptions are listed here.
12 This formula up here is simply a formula that -- this is
13 worked out on a daily basis over a lifetime and so what this
14 formula actually amounts to is incorporating each of the
15 assumptions for each day and then with a computer going
16 through and adding it all up over a lifetime. The factor is
17 the expected lifespan in days. This factor is the age
18 specific amount of soil ingested each day and they have made
19 some estimations of soil ingestion patterns according to age
20 with ingestion of approximately a gram of soil a day between
21 ages 9 and 18 months, 10 grams of soil a day between a year
22 and a half and three and a half years of age and then the
23 amount ingested tends to diminish with age and they have also
24 made estimates of daily deposition of soil on skin with age

1 and those estimates of deposition on scale for the age groups
2 are about the same. And they have made an estimate of the
3 percentage that should be expected to be absorbed through the
4 gastrointestinal tract and they have arrived at a figure of
5 one percent absorption.

6 In other words, you see they have the amount
7 ingested and the amount absorbed. They have the amount that
8 they would estimate to be deposited on skin and the amount
9 that they would expect to be absorbed through the skin. The
10 amount -- this is a symbol for inhalation. This is an
11 estimate of the amount of air exchanged each day. It is
12 known what our respiratory exchange rate is. We probably,
13 with the kind of activity that is going on in here, probably
14 have an exchange rate of something like 10 or 15 cubic meters
15 a day that we breathe in and breathe out. Concentration of
16 dust in the air, and a variable for season. They may be
17 assuming that in the winter months where children are not
18 outside, that that would be zero. So they put in the factor
19 one for the fair weather months and zero for the cold weather
20 months. And they made estimates when they did the ultimate
21 calculation based on one part per billion and on a hundred
22 parts per billion. These are the doses that they estimated.

23 But the main thing we are interested in here are
24 the considerations that they brought into the calculation.

1 They make the point again that they are assuming uniform
2 contamination across the entire area.

3 Q. Doctor Dost, what did they assume would be the
4 primary route of exposure?

5 A. By ingestion of soil and primarily at the age range
6 of one and a half to three and a half years where they assume
7 10 grams of soil taken in a day.

8 Q. 10 grams per day?

9 A. Yes.

10 Q. And what about the routes of dermal exposure or
11 inhalation?

12 A. They came to the conclusion that inhalation
13 exposure was not significant and they made a note of that but
14 they did not include inhalation as part of the calculation
15 because the factor is so small that it made no contribution
16 to the ultimate estimated dose.

17 Q. And did they look at the time of exposure? In
18 other words, the length of time?

19 A. Well, they looked at -- they set this formula up to
20 deal with the entire lifetime. In other words, when they
21 arrived at an estimated dose, they are making the assumption
22 of a total lifetime exposure. In other words, if I were to
23 live from birth to death in one site and the assumption is
24 made that the contamination level remains constant throughout

1 that time.

2 Q. And did they build in any kind of a safety factor?

3 A. Well, they used, they used the safety factor or the
4 no effect level that is currently accepted by the Food and
5 Drug Administration as well as CDC of a no effect dose at one
6 nanogram per kilogram and then, of course, they find that
7 they have -- well, all right. The average daily TCDD dose to
8 an individual over a 70 year lifetime would be 44.6
9 picograms. Now, we are talking about a no effect level at
10 one nanogram per kilogram per day or 70 nanograms per person,
11 approximately, 50 to 70 kilogram person, it would be 50
12 nanograms and since that is a number that is similar to 44 --
13 That is a thousand fold difference. In other words, 44.6
14 picograms is 1,000 fold less than 45 or, yeah, 50 nanograms.

15 Q. Now, what about the dose response analysis and
16 assumptions for promoters versus carcinogens? What sort of
17 assumptions did they make there?

18 A. Well, they made the assumption -- you understand
19 that this is a document intended for regulatory use. It is
20 the practice in the regulatory context because the regulatory
21 context looks at events that might be in the future, not
22 events that are in the past. And even though they make the
23 statement in the document that TCDD is apparently a promoter,
24 they use the assumption that it is a complete carcinogen. In

1 other words, they used an assumption that does not include a
2 threshold. In other words, the dose response relationship is
3 essentially linear down to zero dose which is the custom in
4 any kind of regulatory context at the present time.

5 Q. All right. Now, how does that square with what the
6 studies show with respect to whether or not there is a
7 threshold?

8 A. In this document itself, they make the point that
9 it is, I can't remember their exact language, it is possible
10 or it is likely that the dose response relationship with
11 respect to a promoter and, therefore, TCDD may be non
12 linear. That is, that it may come down to a threshold or
13 have some other configuration that approaches a threshold and
14 what they do is acknowledge that along with the other
15 assumptions that they make and the other estimates that they
16 make, they are setting up a highly conservative model from
17 which to judge the potential hazard of a one part per billion
18 contamination.

19 Q. And as a matter of fact, if you look at the Exhibit
20 1255 there, I direct your attention to the second page of the
21 exhibit in the middle of that first paragraph.

22 A. These calculations assume that a linear dose
23 response relationship exists for carcinogens such as TCDD
24 that based on current evidence are thought to be primarily

1 promoters. However, the dose response curve for promoters
2 may not be linear causing an over-estimated risk. Is that
3 what you are referring to?

4 Q. Yes. Now, Doctor Dost, have you been to Sturgeon?

5 A. Yes, I have been to Sturgeon.

6 Q. And how did you go?

7 A. I believe it was in late July or early August. I
8 only remember that it was very, very warm when I was there.

9 Q. And what did you do while you were there? Where
10 did you go?

11 A. Well, I walked the trackage from the, actually from
12 the middle of Sturgeon through what is it Ogden Street, Wentz
13 Street and on clear out to the intersection past the site
14 where the tank car came to rest and then back. Inspected the
15 area so that I could get an understanding of the topography
16 and the kind of land that was adjacent. Visualize the
17 treatment facilities that were set up and the excavations
18 that were done and so forth.

19 Q. Now, at the time you were there, of course, none of
20 that equipment was there?

21 A. Everything was gone.

22 Q. Did you have photographs with you from which you
23 could visualize where the things were?

24 A. I had studied photographs very extensively before I

1 went. I took a couple along so that I could keep oriented
2 but, yes.

3 Q. Are you familiar with the smell of chlorinated
4 phenols, sir?

5 A. Yes, I am afraid I am.

6 Q. And were you able to detect the smell of
7 chlorinated phenols when you were there on that hot day?

8 A. No. I couldn't smell them.

9 Q. And where else did you go besides walking up and
10 down the track?

11 A. Well, we walked in some parts of the town, entered
12 a couple of stores. We were debating whether to have lunch
13 there or whether to go someplace else for lunch. Bought a
14 soft drink. Drove throughout the town. Followed the course,
15 most of the course of the ditch that leads from the, from
16 about the point where the French drain ended and, in general,
17 toured throughout the town. Walked some of it, drove through
18 some of it and then went out into some of the surrounding
19 agricultural area to get a general sense of the nature of the
20 community.

21 Q. Now, with respect to Sturgeon, sir, and your
22 understanding of this incident, was residential soil
23 contaminated by the spill in Sturgeon?

24 A. I don't believe so. I can't see how it would

1 become contaminated.

2 Q. Now, what was the area that was contaminated by the
3 spill?

4 A. The area that was primarily contaminated by the
5 spill is an area just outside the town.

6 MR. CARR: I object unless the witness states the
7 basis for his knowledge of the area that was contaminated by
8 the spill.

9 THE COURT: Objection is sustained.

10 MR. CARR: Unless counsel gives it to him in a
11 hypothetical. I am sure this witness has no knowledge of his
12 own.

13 THE COURT: Objection is sustained.

14 Q. Doctor Dost, if you were to assume that the car
15 came to rest on the main line track adjacent to the Triple C
16 Construction property there at Sturgeon. Did you see the
17 Triple C Construction property there?

18 A. I did, yes.

19 Q. And if you assume that the car came to rest there,
20 sir, and that the derailment began near Ogden and Wentz
21 Streets and the car proceeded west from there along the track
22 until it came to rest?

23 A. Yes.

24 Q. Now, what was the area that received, if the car

1 came to rest there, the area that received the primarily
2 contamination?

3 MR. CARR: Your Honor, I object to that. Counsel
4 hasn't given the witness any facts yet. The witness can't
5 answer a hypothetical, give a factual answer to a
6 hypothetical.

7 THE COURT: I think there is some lacking in that
8 question, too. Could you rephrase it and ask more. I will
9 give you that opportunity. Objection is sustained.

10 Q. Doctor Dost, can you tell me which of the
11 assumptions upon which the CDC estimate was based in your
12 opinion do not apply to the Sturgeon situation?

13 MR. CARR: Your Honor, I object until the witness is
14 given the facts to make an opinion on it. Right now he has
15 been given no facts except that a tank car ran through
16 Sturgeon.

17 THE COURT: Objection is sustained.

18 Q. Now, what is the age, Doctor, at which one would
19 expect ingestion of soil that might be contaminated? What is
20 the age at which that is expected to occur?

21 A. Well, according to the CDC and its consultants, the
22 age at which the maximum amount of soil might be ingested
23 would be between the ages of one and a half and three and a
24 half. Somewhat less prior to that time and the amount would

1 diminish until by the age of five the estimated intake is on
2 the order of 100 milligrams a day of soil, dirt.

3 Q. All right. Now, how is that according to the CDC
4 assumption, how is that soil ingested? How does that occur?

5 A. Well, just because children do not eat much dirt
6 purposefully but children are not that careful about the way
7 they handle toys and they tend to take a lot of soil in just
8 through carelessness.

9 Q. Is that in the area where they play?

10 A. Primarily in the area where they are playing, where
11 they are spending most of their time, yes.

12 Q. And where do children who are ages one and a half
13 to three and a half years spend most of their time, according
14 to the CDC estimate?

15 A. Well, I would assume that at times when they are
16 not under immediate and direct supervision, they are in their
17 own yard and in their own home.

18 Q. And it is at that location that the CDC is assuming
19 that this ingestion of dirt is going to occur?

20 A. That is correct.

21 Q. Does the railroad right-of-way at Sturgeon that you
22 observed run through the yards of any of the people that live
23 in the town?

24 A. I believe there is one residence through which or

1 near which the railroad right-of-way runs. It doesn't run
2 through any yards.

3 Q. Do you have a conclusion, sir, as to -- do you have
4 an opinion as to whether the one part per billion level of
5 concern established by CDC in this document would be
6 applicable to Sturgeon?

7 MR. CARR: Your Honor, I object unless it is based
8 simply upon what the doctor has yet to suggest to and not any
9 other consideration because no other facts have been given to
10 Doctor Dost.

11 THE COURT: With that caveat, you may answer the
12 question, Doctor Dost. I think that is properly brought
13 out.

14 A. In my opinion, there is no relation to an exposure
15 that might take place at any point on a railroad right-of-way
16 and an exposure in a residential yard and I don't think that
17 the CDC estimate is applicable to the situation in Sturgeon.

18 Q. Doctor Dost, with respect to dioxin isomers other
19 than 2,3,7,8 TCDD, is there any literature that treats the
20 toxicity of those other types of dioxin isomers?

21 A. Yes. There are some. There is some research in
22 this area.

23 Q. How do the other dioxin isomers relate in terms of
24 toxicity vis-a-vis 2,3,7,8 TCDD?

1 MR. CARR: Your Honor, I object unless he
2 identifies the source of his knowledge at this point and
3 cites the literature.

4 THE COURT: Objection sustained. Can you rephrase
5 the question.

6 Q. What literature are you referring to, Doctor Dost?

7 A. Quite a few years ago Doctor Swetz did a
8 comparative lethality study on TCDD and other chlorodioxins.

9 MR. CARR: Can you spell his name?

10 A. S-c-w-e-t-z. There have been studies of the
11 ability of TCDD to induce and others as a comparative study
12 TCDD and other isomers to induce AHH in cultured cells as
13 well.

14 Q. And who has done that work?

15 A. Primarily a group including Doctor Bradlaw, Doctor
16 Casterline.

17 Q. And what do these studies demonstrate with respect
18 to the toxicity of the other dioxins?

19 MR. CARR: Your Honor, to my knowledge we have not
20 been given these studies. The witness hasn't referred to
21 them and we didn't -- It is not in the document that he has
22 given us.

23 MR. HEINEMAN: I certainly believe you have been.
24 I don't know that I have that sheet here. Which one is it

1 that you don't have, Mr. Carr?

2 MR. CARR: I have got nothing that refers to Scwetz
3 at all. I have got nothing that refers to Bradlaw at all.

4 THE COURT: Gentlemen, it is almost noon. Why
5 don't you resolve this over the lunch break and we will start
6 again at 1:15. Court is in recess for lunch. We will resume
7 again at 1:15. I would remind you that you are not to
8 discuss this matter among yourselves, with anyone outside the
9 jury panel or as of yet form any opinions or conclusions
10 about the matters on trial. Court is in recess.

11 COURT RECESSED:

12 (The following proceedings were had in the hearing
13 and presence of the jury)

14 FRANK DOST

15 having resumed the witness stand, being previously sworn,
16 testified further as follows:

17 MR. HEINEMAN: Your Honor, may counsel approach the
18 bench for just a minute please.

19 THE COURT: Sure.

20 (Bench conference had out of the hearing of the
21 jury.)

22 MR. HEINEMAN: Right before the lunch break, Judge,
23 Mr. Carr had raised an objection with respect to having
24 testimony on the, with respect to the paper by Bradlaw.

1 THE COURT: Bradlaw and someone else.

2 MR. CARR: The Swetz paper we have. There were
3 two papers. We have the Swetz paper.

4 MR. HEINEMAN: Okay. My understanding is that both
5 papers were in the Carnow bibliography which was provided to
6 Mr. Carr a long time ago but it is also my understanding that
7 in the list of papers which Doctor Dost was going to rely on,
8 the Swetz paper was mentioned by the Bradlaw was not.

9 MR. CARR: That is correct.

10 MR. HEINEMAN: And, therefore, I am not going to
11 have him testify to anything involving the Bradlaw paper so
12 the questions.

13 MR. CARR: You have a copy of the Bradlaw paper
14 here?

15 MR. HEINEMAN: I don't have one here. No. I don't
16 think so. I don't think so. I am not going to ask him
17 anything about it.

18 THE COURT: About Bradlaw?

19 MR. HEINEMAN: No. Because I think Mr. Carr has a
20 legitimate objection.

21 MR. CARR: And it wasn't listed.

22 THE COURT: But you are going to ask him about
23 Swetz?

24 MR. HEINEMAN: Yes.

1 THE COURT: And since you have it, there is no
2 objection?

3 MR. CARR: Correct.

4 (The following proceedings were had in the hearing
5 and presence of the jury)

6 DIRECT EXAMINATION

7 By

8 MR. KENNETH R. HEINEMAN.

9 Q. Doctor Dost, before we broke for lunch, we were
10 talking about the work of a Doctor Scwetz and with respect to
11 investigating the lethality of certain isomers of dioxin. Do
12 you recall that, sir?

13 A. Yes.

14 Q. All right. And in connection with Doctor Scwetz's
15 work --

16 MR. CARR: Counsel, would you make it clear that he
17 mentioned other reports before lunch and that you are not
18 going to ask him about it in the record, just the Scwetz?

19 MR. HEINEMAN: I am not going to ask you about the
20 Bradlaw, just the Scwetz.

21 Q. With respect to the other isomers of dioxin, what
22 does the Scwetz work indicate as to the relative toxicity?

23 A. Well, the TCDD is, of course, the most toxic. The
24 other four chlorine isomers that they tested were much less

1 toxic on a comparative -- in fact, they were so much less
2 toxic that they had trouble getting good quantitation. Their
3 estimate of the lethality was that it required in excess of
4 100 milligrams per kilogram which would be 100,000 micrograms
5 while in the rat whereas TCDD is lethal at a dose on the
6 order of 20 to 30 micrograms per kilogram in the rat.

7 Q. As opposed to 100,000?

8 A. As opposed to 100,000 for other four chloro
9 isomers. The two chloro isomers, 2,7 and 2,8 are another
10 order of magnitude, less toxic still. On the order of what
11 would amount to a million micrograms per kilogram.

12 Q. Now, when you are talking about the other four
13 chloro isomers, are you including 1,3,6,8?

14 A. 1,3,6,8, 1,3,7,9. These are four chloro,
15 tetrachlorodioxins with the chlorines located other, at other
16 places than on the outside corners. We could have two of
17 them, for example, in those positions but if the others are
18 not, the toxicity drops precipitously.

19 Q. Now, what about the hexas and octas?

20 A. They are -- in part, it depends on the location of
21 the chlorines, on the hexas, but the most toxic is perhaps 10
22 to 15 fold less toxic than TCDD.

23 Q. And with the octas, sir?

24 A. The octas are relatively innocuous. They are not

1 very toxic at all.

2 Q. Now, sir, I would like to talk to you about phenol
3 and chlorinated phenols. Would you tell the jury what phenol
4 is? What are its physical characteristics?

5 A. Phenol is a much simpler compound than is TCDD. It
6 is -- at room temperature it is a clear crystalline
7 material. If it is a little bit contaminated, it may be
8 slightly pink in color but it is basically a clear
9 crystalline substance that has a vapor pressure of about a
10 third of a millimeter of mercury which means that it has some
11 volatility but it is not highly volatile and it has a very
12 strong odor.

13 Q. Now, speaking of the odor, Doctor Dost, would you
14 define the difference for us between odor threshold and TLV?

15 A. An odor threshold is the concentration in the
16 atmosphere that can be detected by humans and this is
17 determined by using a so-called odor panel. In the same way
18 the taste panels are used for judging the taste of food
19 products in which a group of people are individually exposed
20 to varying concentrations of the substance and when they can
21 first tell that it is in the air, then they identify that
22 concentration. The TLV or the threshold limit value is a
23 factor that is used in industrial hygiene to identify the
24 level of a compound that is acceptable for workday exposure

1 in the workplace.

2 Q. What is the odor threshold for phenol?

3 A. For phenol it is about on the order of five
4 hundredths of a part per million. .05 parts per million.

5 Q. Which would be how many parts per billion?

6 A. That would be, that would be 50 parts per billion.

7 Q. And what is the threshold limit value in the
8 workplace for --

9 A. The threshold limit value in the workplace for
10 phenol is five parts per million.

11 Q. Which would be how many times higher?

12 A. It would be 100 times higher than the odor
13 threshold.

14 Q. All right. Now, do you recall the testimony of
15 Doctor Silbergeld that phenols can be detected in the air
16 between 1 to 10 parts per million and OSHA recommended a TLV
17 for phenol at the level you can detect or on the order of one
18 part per million? Do you recall that testimony, sir?

19 A. Yes, I do remember that.

20 Q. Do you agree with that?

21 A. No, sir, I do not.

22 Q. All right. And on what basis do you disagree?

23 A. Well, really on the basis that I have just stated.
24 That the odor threshold is substantially lower than that.

1 The odor threshold is at .05 parts per million which is 20
2 times less than one part per million and that the OSHA TLV
3 which was last reviewed in, I believe, 1976 is at five parts
4 per million.

5 Q. So, would it be true, sir, that if you can smell
6 phenol in the air, then there is clearly more there than
7 there should be according to the OSHA standard?

8 A. Not according to the OSHA standard, no, sir.

9 Q. Now, what happens to phenol in the body, sir?

10 A. Well, phenol is absorbed very readily. It is
11 absorbed when its contact is by the digestive tract. Phenol
12 vapor can be absorbed even across the skin. In fact, the TLV
13 identifies that fact by stating that phenol exposure is not
14 limited to the respiratory exposure but also to vapor
15 exposure across the skin. Phenol is not held in the body for
16 a very long time. It is a non specific compound. It doesn't
17 have preferential sites to which it moves. It is
18 sufficiently water soluble; that substantial amounts are
19 excreted without change but phenol also goes through this
20 process of glucuronidation which I have discussed in which a
21 soluble molecule is attached to it and it then goes out in
22 the bile or perhaps in the urine.

23 Q. Is that through metabolism?

24 A. In a sense. It is metabolism in the sense that the

1 compound is changed. We don't take something off of the
2 compound except the one hydrogen off of that hydroxyl group
3 and exchange it and put on another more soluble group but the
4 ring is not broken up. The basic structure of the compound
5 is not changed. If we were to subject that glucuronide to a
6 glucuronidase enzyme later on, you would get phenol back out
7 of the system.

8 Q. Does the human body in its normal state contain
9 some phenol?

10 A. Yes. As a matter of fact, it probably arises
11 primarily from the activity of bacteria in the digestive
12 tract as they interact, as they metabolize certain amino
13 acids, tryosine. Some of the aromatic amino acids are broken
14 up to form phenol.

15 Q. And where do those materials come from, sir?

16 A. Well, those are normal constituents of the diet.

17 Q. Now, is it normal to find either in the animal or
18 in the human certain constituents of phenol in the urine?

19 A. Yes. It is not -- anytime that one is looking for
20 baseline levels of phenol, if you were looking at an exposure
21 to extrinsic, that is phenol coming from the outside, you
22 have to take a baseline level to learn what the level of
23 phenol in the urine already is.

24 Q. Well, Doctor, what are the toxic properties of

1 phenol?

2 A. Phenol is what sometimes is called a protoplasmic
3 poison because it does haven't any specific effect. At any
4 point where the concentration becomes sufficient, it simply
5 causes damage. It will cause damage in the kidneys if a
6 large amount is taken in; the liver, almost any organ where
7 the circulation is substantial. On the skin it is a serious,
8 it is a severe caustic. Phenol will burn the skin. If
9 someone was to ingest phenol, it would devastate the upper
10 digestive tract. The mouth, the esophagus and so forth.

11 Q. It can cause death, can it not?

12 A. It certainly can. It is a highly toxic substance.

13 Q. Does it have any genetic potency?

14 A. It doesn't appear to have an appreciable ability to
15 cause genetic change. It is a rather non specific poison
16 because it is so reactive, I suppose, that it is imaginable
17 that it could perhaps cause some damage. I, at the moment,
18 do not know of specific tests that show any positive results
19 of it in genetic studies.

20 Q. What are the levels of phenol that are found to be
21 required to cause some sort of biological reaction?

22 A. I would say that a dose of 20 or 30 million grams
23 per kilogram would cause toxicity. The lethality of the
24 phenols runs in between two and 400 milligrams per kilogram

1 depending on the species. Some are somewhat higher.

2 Q. Is the TLV, I assume, lower than any level that
3 would cause any --

4 A. Heavens yes. That is the whole idea of
5 establishing industrial hygiene limits with any substance.
6 TLVs are set for all substances that are encountered in the
7 industrial environment, at least nearly all substances.

8 Q. Is there any evidence, sir, that chronic phenol
9 exposure below the odor level is harmful, toxic?

10 A. I know of no evidence.

11 Q. As a matter of fact, the odor level is far below
12 the TLV, isn't that correct?

13 A. The odor level is below the TLV and it is-- because
14 the odor of phenol is so sharp, there is very little question
15 when one is in its presence.

16 Q. So that if Doctor Silbergeld testified, sir, that
17 the toxic effects from chronic phenol exposure below the
18 odor, can arise below the odor level --

19 MR. CARR: What page is that and what day?

20 MR. HEINEMAN: April 16, page five.

21 Q. If she had testified, sir, that chronic exposure at
22 levels below the detection by smell can result in toxic
23 effects, would you agree or disagree with that statement?

24 A. I cannot agree with that.

1 Q. As a matter of fact, is there phenol found in the
2 body all the time?

3 A. Yes.

4 Q. Now, there is evidence, sir, in this case. There
5 is testimony that people tested the levels for phenol at
6 Sturgeon. In fact, the EPA measured phenol in the air at
7 Sturgeon. I would like you to assume that and assume that
8 there was never a finding by either EPA or Monsanto of phenol
9 above the threshold limit value. Could you assume that, sir?

10 A. Uh-huh. Yes.

11 Q. I would like you also to assume that there was
12 testimony in the case that people could smell strong phenolic
13 odor as far away as a mile or two from the spill site
14 according to testimony in the case. Will you accept that,
15 please, sir?

16 A. Yes.

17 Q. Can you tell me what it was they would be smelling?

18 A. Well, most of the material that spilled was two
19 chlorophenol and two chlorophenol has a very, very sensitive
20 odor threshold. It has a recognition threshold on the order
21 of 95 parts per trillion. In other words, not only can it be
22 detected but a person who is familiar with that smell can
23 identify it at 95 parts per trillion and that level generally
24 comes quite a bit, at a higher concentration than the

1 detection threshold or the odor threshold and I wouldn't at
2 all be surprised that it could be smelled some distance away
3 because that is a very, very small amount of material.

4 Q. Now, by two chlorophenol, is that the same thing as
5 orthochlorophenol?

6 A. That is orthochlorophenol.

7 Q. Now, I would like you to assume that Doctor
8 Silbergeld testified at page 14 on April 16th that the
9 process of metabolizing phenol can cause damage to the liver
10 and kidney. Would you assume that, sir?

11 A. All right.

12 Q. Do you agree with that statement?

13 A. Well, when the concentration of phenol in the body
14 becomes high enough, it is going to damage the liver and the
15 kidney but it has nothing to do with the process of
16 metabolism. It has to do with the fact that the
17 concentration is so high that it is causing cellular
18 destruction.

19 Q. So that if the concentration of phenol in the body
20 is below that at which, for example, skin destruction or
21 tissue destruction would occur by the mere contact, would the
22 fact that metabolizing it cause any toxicity to either the
23 liver or the kidney?

24 A. No. There is a dose response with this sort of

1 thing just like every other kind of chemical impact and below
2 the level where there is actually being cellular destruction,
3 there is going to be a variety of toxicological injury but
4 this has nothing to do with the metabolism of the compounds.
5 It has to do with the reaction of the compound in the tissues
6 as it exists as the phenol. The process of metabolism to the
7 extent that it takes place is really a means of getting rid
8 of the material. And at lower levels, the mammal, humans and
9 other animals are very, very efficient at disposing of it.

10 Q. Well, sir, let me ask you if you recall the
11 testimony of Doctor Silbergeld on April 16, page four, with
12 respect to the toxic effects of phenol on the liver being
13 related to the removal of chlorine from the phenol ring
14 producing what is called a free chlorine radical. Do you
15 remember --

16 A. Yes.

17 Q. That testimony, sir? Do you agree with that?

18 A. No, I do not.

19 Q. And can you tell us why?

20 A. There is no evidence at all that chlorine is
21 metabolized by any mechanism that removes chlorine from the
22 ring. Furthermore, the idea of a free chlorine radical as an
23 entity in the toxicity of a chlorinated compound is really
24 not sound because chlorine, when it is removed from organic

1 ring structures, is removed as a chloride ion and it is not
2 highly reactive. Our body is saturated with chloride ion.
3 We are full of sodium ions and chloride ions. We can't
4 survive without it. If it were a highly reactive species.
5 It would be no more reactive than the multitude of active
6 oxygen and peroxide and so forth I believe that I was
7 discussing yesterday as agents that are, that occasionally
8 cause damage to DNA and there are multitudes of scavenging
9 mechanisms that straps such substances and removes them from
10 any activity. But basically the best argument is very simply
11 that that kind of reaction does not occur with a
12 chlorophenol.

13 Q. Now, Doctor Dost, you suggested that sodium,
14 charged sodium and chloride ions are necessary to our
15 existence?

16 A. Well, of course. When I say charged, we are simply
17 talking about, about ionized sodium, ionized chloride. That
18 is the state that sodium chloride is in in solution.

19 Q. What is sodium chloride?

20 A. It is salt, just like table salt.

21 Q. And so that negatively charged chlorine atoms and
22 positively charged sodium atoms are in the body all the time?

23 A. We won't call them atoms. They are ions because
24 they have had a change in their electrical charge but they

1 are not, they are not destructive entities. They don't go
2 around having violent interactions with other components of
3 the organism.

4 Q. Doctor Dost, would the fact that people were able
5 to smell orthochlorophenol at Sturgeon mean that 2,3,7,8 TCDD
6 was present in the atmosphere?

7 A. Certainly not.

8 Q. Why do you say that?

9 A. Orthochlorophenol is quite volatile. It also has a
10 very low odor threshold. TCDD is virtually not volatile.
11 Its volatility is so low as to be considered essentially nil.

12 Q. To your knowledge, sir, has TCDD ever been measured
13 in ambient atmosphere anywhere in the world?

14 A. I know of no case where it has been measured in the
15 atmosphere as a gas.

16 Q. Are there any other factors which would lead you to
17 believe that TCDD would not be present in the atmosphere
18 other than what you have already told us?

19 A. Well, TCDD has a very strong tendency to bind to
20 anything that it comes in contact with and that would inhibit
21 its ability to move. It also is subject to degradation by
22 light.

23 Q. All right, sir. Have there been studies done on
24 the toxicity of 2,4 dichlorophenol?

1 A. Yes. Yes.

2 Q. And what do they demonstrate, sir?

3 A. They demonstrate again that the compound is similar
4 to the other chlorophenols in that the toxicity is relatively
5 non specific. That is, it attacks when it reaches
6 concentration that is sufficient to simply interact directly
7 with cells. It is rather quickly excreted. Half times are
8 very short. It appears to have very little reproductive or
9 immunological effect. It is like all chemicals, fetotoxic in
10 sufficient concentrations. In other words, if 2,4
11 dichlorophenol is given to a pregnant animal, not only the
12 parents but the offspring would be expected to be intoxicated
13 at sufficient doses.

14 Q. You say in sufficient doses. Does it have a no
15 observed effect level?

16 A. It appears to, yes.

17 Q. All right. Doctor Dost, I would like to ask you a
18 hypothetical question, sir. And I want you to assume, if you
19 would, that on the night of January 10, 1979, a tank car
20 derailed while moving west through Sturgeon, Missouri.
21 Assume that this tank car spilled approximately 19,000
22 gallons of orthochlorophenol-crude, a mixture of phenol and
23 various chlorinated phenols, and that this mixture also
24 contained between .6 and 45 parts per billion of 2,3,7,8

1 tetrachlorodibenzo-para-dioxin which would be no more than 81
2 percent of a teaspoon in this 19,000 gallons of chemicals.

3 Assume also that the spillage involved the main
4 line track, the passing track and a seven foot wide walkway
5 between the two tracks. Further assume that this tank car
6 traveled 2700 feet after it derailed, coming to rest beyond
7 the city limits of Sturgeon; that there were only scattered
8 splatters and splashes of chemical on the ground, along the
9 passing track the first 2,400 feet past the point of
10 derailment and that the major spill area was 240 feet long at
11 the extreme western end of the spill area where the tank car
12 came to rest.

13 Assume that dioxin has a low volatility, 16 billion
14 times less than water at 77 degrees Fahrenheit, so that it
15 does not tend to become airborne or turn into gas.

16 Assume that the inhabitants of Sturgeon were
17 evacuated the night of January 10, 1979, but were notified
18 they could return to Sturgeon January 12, 1979, after the EPA
19 determined from air samples taken by the EPA and by Monsanto
20 that no health hazard existed from breathing the air at
21 Sturgeon. Assume that the air in the town of Sturgeon was
22 sampled for four to six months starting in April of 1979 and
23 that no levels in excess of one part per million of any of
24 the spilled chemicals were ever detected in the air in the

1 town of Sturgeon.

2 Assume that the railroad retained a contractor to
3 clean up the site of the spill under the direction of the
4 EPA. Assume that the contractor dispatched a team of workers
5 who arrived at Sturgeon at nine o'clock a.m. the morning
6 after the spill. Assume that the first operation in
7 connection with cleanup of the track they performed was
8 cribbing or removing material from between the ties. Assume
9 that all the material between the ties and the main track was
10 removed to a depth of six to eight inches below the bottom of
11 the ties as well as all material between the end of the
12 passing track ties north of the rails. In addition, assume
13 that all material between the passing track ties was removed
14 to a depth of eight to ten inches in the western 150 to 200
15 feet of the spill area where the surface of the snow and the
16 ballast of the passing track had been visibly discolored by
17 the spilled chemical.

18 Assume that by January 26, 1979, all of this
19 material had been removed from the tracks and transferred by
20 gondola car to a staging area south of the tracks which had
21 been scraped and surrounded by a wall of soil to contain the
22 material. Further assume that no dust was kicked up either
23 by the cribbing operation or by the transfer of this material
24 to the staging area. Assume that this material removed from

1 between the ties was placed in two piles in this staging area
2 and that the cleanup workers were instructed to keep any
3 contaminated material confined to these piles.

4 Assume that this material never thawed while it was
5 in the piles and that there was no dust blown off the piles
6 as there were no particles small enough to be picked up by
7 the wind. Assume that a backhoe lifted soil and ballast out
8 of these piles and loaded it into 55 gallon drums. The
9 larger or east pile was covered with plastic as the west pile
10 was loaded into the drums.

11 During the loading of the barrels, there was no
12 dust picked up by the wind. Workers then leveled off the
13 material by hand to the top of the barrel and secured the
14 lid. Assume that after the lid was securely fastened, the
15 workers rolled the barrels away from the contaminated area so
16 that the scatbacks which picked up the barrels would not
17 drive over any contaminated material. Assume that as
18 material was loaded from the piles into the barrels, the
19 backhoe operator would scoop up any material which was no
20 longer on the pile back onto the pile so that the backhoe
21 would not be tracking through any contaminated material.

22 Assume that after the barrels were rolled away from
23 the contaminated area, they were loaded by scatbacks onto
24 flatbed trailers which were then pulled to the north side of

1 the tracks where the barrels were unloaded for temporary
2 storage. Assume that it is possible that less than a cubic
3 yard of contaminated material was carried out of the staging
4 area on the wheels of the scatbacks or flatbed trailers.
5 Assume that the barrels were reloaded on the north side of
6 the tracks into enclosed tractor trailer trucks to be
7 transported out of Sturgeon. And that no drums left the site
8 in anything other than enclosed tractor trailer trucks.

9 Assume that by March 1, 1979, 4,500 drums of
10 material had been removed from Sturgeon. Assume that after
11 the cribbing operation was completed, a ditch two and a half
12 feet wide and four feet deep was dug between the passing
13 track and the main track from Ogden Street to the place the
14 tank car came to rest. Assume that additional material was
15 also removed onto the main track by an undercutter machine.
16 Assume that this machine removed the ballast and earth
17 beneath the main track to a depth of 46 inches in the 200 to
18 240 feet of track where the most chemical was spilled and to
19 a depth of two feet for the rest of the main track.

20 Assume also that some dirt was removed from the
21 ditches around where the tank car came to rest. Assume that
22 all of this material was loaded into trucks previously lined
23 with sheets of plastic. Assume that in order to prevent any
24 spillage of soil, the trucks were not filled to the top, the

1 plastic was folded over the top of the soil and a heavy
2 canvas tarp was secured over the top of the truck and no
3 truck left the spill site without a plastic liner and a
4 canvas tarp. Assume that by March 1, 1979, 2,855.3 cubic
5 yards of soil had been removed from Sturgeon in these
6 trucks.

7 Assume that the cleanup workers spent the night in
8 towns other than Sturgeon and only went into the town of
9 Sturgeon for lunch with the exception of a couple of workers
10 who went into town for hardware supplies on days when they
11 were not working in the contaminated material. Assume that
12 the workers always went to Daisy's Cafe for lunch. Assume
13 that when the workers arrived in the morning, they put on
14 protective PVC rain suits and rubber gloves kept on site and
15 that when the workers left the site for lunch at Daisy's or
16 at the end of the day, they always removed their protective
17 suits. Assume that when the workers were at the western end
18 of the spill site, they always either brushed off their boots
19 before leaving the site with a dry brush or scrub them with a
20 brush and soap and water. And that even when the workers
21 worked at the less contaminated eastern end of the site, some
22 still scrubbed their boots before leaving the site.

23 Assume that throughout the January and February
24 cleanup, the weather was nearly always below freezing.

1 Sometimes as low as 20 degrees below zero and that this was
2 too cold for soil and other material to stick to boots or
3 tires. Assume that the only place where mud was generated
4 during this period was in a non contaminated area. Assume
5 that no cleanup worker ever testified that he saw any mud
6 tracked anywhere by the cleanup workers or that anyone ever
7 complained that the workers were tracking mud into Daisy's or
8 anyplace else.

9 Assume also that one of the duties of the EPA on-
10 site cleanup coordinator was to see if any chemicals were
11 tracked away from the cleanup site and that he never observed
12 any chemical being tracked out of the controlled site area by
13 any equipment or personnel. Assume that there were banner
14 guards erected at the Ogden Street crossing to prevent
15 unauthorized people from getting into the site and that no
16 unauthorized people were seen on the site. In addition,
17 assume that the railroad posted railroad security guards
18 during the weeks following the derailment at the Triple C
19 driveway to restrain on-lookers, spectators and townspeople
20 that did not have any business being there from driving into
21 the Triple C driveway.

22 Assume that Monsanto informed the EPA on February
23 9, 1979 that it had confirmed the presence of
24 tetrachlorodibenzo-para-dioxin in the spilled material at a

1 level of approximately 37 parts per billion and that EPA
2 determined at that time in consultation with outside
3 toxicologists that even if the dioxin present was the most
4 toxic form of TCDD, no increased endangerment to the towns-
5 people was expected as a result of this information and that
6 the primary problem was exposure to the phenolic compound.

7 Assume the ponds south of the railroad tracks were
8 found in March of 1979 to be discolored in the same manner
9 that water known to contain phenol compounds is discolored
10 and that these ponds were found to contain orthochlorophenol
11 and phenol compounds. Assume further that discolored water
12 was also observed in Saling Creek when it overflowed its
13 banks during the latter days of April. Assume also that
14 dioxin has a very low solubility in water and, therefore,
15 would not tend to migrate with this water unlike phenol and
16 chlorinated phenols which are water soluble.

17 In this regard, assume that a sample of water
18 obtained by Doctor Gary Osweiler on April 4, 1979 from a
19 ditch north of the main track at the spill site was analyzed
20 by Doctor Matthew Zabik and found to contain over 15 percent
21 phenol and chlorinated phenols but only 14 parts per trillion
22 TCDD with no 2,3,7,8 tetrachlorodibenzo-para-dioxin present.

23 Assume further that dioxin has a strong attraction
24 to soil and would tend to bind to soil rather than to become

1 airborne or to migrate with water flowing through the soil
2 and that, therefore, the movement of phenol and dioxin
3 through soil will not be similar.

4 Assume that the railroad hired a second cleanup
5 contractor which arrived in Sturgeon during the first week of
6 April of 1979 and worked at the site until June of 1980.
7 Assume that this contractor was originally retained to treat
8 the water in the Kemner pond, to reduce the level of
9 contaminants to a level set by EPA at less than 100 parts per
10 billion. And to reduce the level of contaminants in soil in
11 the area to a level set by EPA of 200 parts per million.

12 Assume that EPA also subsequently established a
13 criteria of 60 parts per billion for runoff water from the
14 spill site into the Kemner pond. In May of 1979 assume that
15 this contractor learned from Monsanto that 37 parts per
16 billion tetrachlorodibenzo-para-dioxin had been in the
17 spilled material and that EPA had labeled the entire amount
18 as 2,3,7,8. Assume that this contractor discussed this
19 information and determined that no changes need be made in
20 the cleanup methods used as a result of those discussions and
21 that EPA also did not require any change in cleanup methods
22 as a result of any information about dioxin content and the
23 spilled material. Assume that this cleanup contractor
24 determined that the source of the water contamination in the

1 Kemner pond was the western most 220 feet of the spill site
2 which lay outside the city limit of Sturgeon.

3 Assume further that the second cleanup contractor
4 contained the runoff from this area to channel it into the
5 Kemner pond, installed a carbon filtration system in the pond
6 south of the railroad tracks and installed an underground
7 pressure injection system to flush the chemicals in the
8 railroad bed into the pond for treatment. Assume that this
9 cleanup contractor also observed contamination in Saling
10 Creek on April 27, 1979, and traced the source of that
11 contamination to a French drain located under the railroad
12 tracks in the eastern portion of the spill site.

13 Assume that the site manager for the cleanup
14 contractor testified that the spilled chemical did not move
15 into the French drain the night of the spill because of the
16 ground frost but instead was carried into the French drain
17 with the water several months later. Assume that that same
18 day the contractor constructed dams to prevent discharges
19 from the French drain from north into the town of Sturgeon or
20 into Saling Creek along the railroad right-of-way. Assume
21 that the contractor treated all the water collected behind
22 these dams before it was discharged into Saling Creek and
23 that a permanent drain pipe was installed to route water from
24 the French drain onto the railroad tracks to Saling Creek

1 where it was filtered by a second carbon filtration system
2 before it entered the stream.

3 Assume that in all 106 water samples analyzed by
4 the contractor in the Sturgeon area during a period from May
5 22, 1980 through June 23, 1980, less than 40 parts per
6 billion of orthochlorophenol were detected. Assume also that
7 the soil around the Kemner pond was sampled in May of 1980
8 and that no levels of contamination above the EPA criteria
9 were detected. Assume also that an outside hydrogeologist
10 investigated the ground water in the Sturgeon area and that
11 as a result of this investigation, the EPA determined that
12 there was no health hazard associated with the ground water
13 at Sturgeon. Also assume that in addition to water treatment
14 as described above, the second cleanup contractor also
15 excavated and removed earth in an area 20 feet wide and 393
16 feet long on the north side of the tracks and ranging from
17 four to 29 feet wide for the same length on the south side of
18 the tracks. These areas were excavated to depths from six
19 feet closest to the track to one foot furthest from the
20 track.

21 Assume that during this excavation the contractor
22 sampled the soil in the excavation area and that whenever a
23 level of contamination higher than the level set by the EPA
24 was found, additional excavation was performed at the

1 location of that sample until subsequent samples showed that
2 the EPA criteria had been met. Assume that all the earth
3 excavated was loaded into dump trucks or trailers lined with
4 plastic and was covered with plastic and a canvas tarp before
5 shipping. Assume that between April 19th and June 25, 1980,
6 the second cleanup contractor removed a total of 1,351.7
7 cubic yards of earth from these areas in this manner.

8 After this earth was excavated, assume that the
9 entire area of excavation was layered with an average of six
10 inches of carbon which is capable of absorbing large
11 quantities of organic material. Above this was placed two
12 separate six inch layers of compacted clay mixed with
13 industrial sealer to keep water from leaking into the
14 excavated area. Above this was placed a 6 to 12 inch layer
15 of clay mixed with top soil which was seeded with grass seed
16 to keep the area from eroding. Assume that the only soil
17 sample taken at Sturgeon would show the presence of
18 tetrachlorodibenzo-para-dioxin was performed in 1983 by EPA
19 on a sample taken February 16, 1979, and showed 65 parts per
20 trillion total tetrachlorodibenzo-para-dioxin. Other
21 analyses performed at the same time showed no dioxin in
22 sediment from cropland along Saling Creek at a detection
23 level of three parts per billion and no dioxin in a well
24 water sample at a detection level of 25 parts per

1 quadrillion.

2 Assume also that the half-life of TCDD and the top
3 two millimeters of soil exposed to ultraviolet light is no
4 more than 36 hours so that at the end of that period of
5 exposure to ultraviolet light, one-half of the TCDD in that
6 layer will have been destroyed and that TCDD will move
7 through soil at a rate of no more than .033 millimeters per
8 day. Further assume that based on these facts it has been
9 calculated that TCDD will take at least 60 to 62 days to move
10 through the top two millimeter layer of soil and that a
11 concentration of 65 parts per trillion at the two millimeter
12 level will have been reduced to a concentration of no more
13 than 625 parts per quintillion by the time it reached the
14 surface.

15 Assuming that all the facts stated above are true,
16 do you have an opinion based upon a reasonable degree of
17 toxicological certainty whether a resident of Sturgeon,
18 Missouri, during the period from January 10, 1979, to the
19 present could have been exposed to any amount of 2,3,7,8
20 tetrachlorodibenzo-para-dioxin sufficient to cause any
21 adverse health effects?

22 A. Yes, I have an opinion.

23 Q. And what is that opinion, sir?

24 A. I don't believe that there is any meaningful

1 possibility of such an effect.

2 Q. Now, Doctor, in your experience with the phenoxy
3 herbicides, did you have occasion to review the presence or
4 potential presence of dioxin in 2,4-D?

5 A. Yes, as a matter of fact.

6 Q. In your review, are you aware of whether or not
7 2,4-D, the herbicide, was ever found to contain 2,3,7,8 TCDD?

8 A. I know of no evidence that it has ever -- that
9 2,3,7,8 TCDD has ever been found in 2,4-D.

10 Q. Was 2,4-D tested for dioxin to the best of your
11 knowledge?

12 A. Yes.

13 Q. By whom and when, approximately?

14 A. In 1980 a Canadian researcher studying Canadian
15 production of 2,4-D found that there were a variety of other
16 chlorodioxins in 2,4-D as well as the 2,7 isomer which a
17 chemist would expect to find in a product containing, that is
18 derived from 2,4 dichlorophenol. That is the dichlorodioxin
19 that would be expected in production of that product. It had
20 not been found previously. When that work was reported, it
21 was reported first in a meeting in Rome and then published in
22 the United States. The EPA carried out a sampling program of
23 commercial sources of 2,4-D and they analyzed some 33 samples
24 as I recall. They found no 2,3,7 TCDD. They found traces of

1 others, one or two other tetras. They found traces of one or
2 two trichlorodioxins. In three of the samples they found
3 small amounts of the 2,7 which was somewhat surprising
4 because we anticipated that they should find it in more
5 samples. They found it in one sample, if my memory serves,
6 at a concentration of 57 parts per billion and in two other
7 samples at levels less than 30 parts per billion.
8 Considering that, the 2,7 DCDD has very, very limited
9 toxicity. Those are not toxicologically significant
10 amounts. That generally is the picture that was found in
11 that EPA sampling.

12 Q. Did the testing include a mean and ester product?

13 A. It is my understanding that both the mean and ester
14 products were tested.

15 Q. To the best of your recollection, sir, was 2,3,7,8
16 TCDD ever found in any of those samples?

17 A. No, it was not.

18 Q. To your knowledge, Doctor Dost, has any scientist
19 or anyone ever reported finding 2,3,7,8 TCDD in 2,4-D?

20 A. I know of no such finding.

21 Q. Do you know what 2,4 dichlorophenol is used for
22 industrially?

23 A. I am sure it has many uses but the use that I am
24 familiar with is as a starting material for the manufacture

1 of the herbicide 2,4 dichlorophenoxyacetic acid, 2,4-D.

2 Q. Doctor Dost, I want you to assume the following
3 facts. That 2,4 dichlorophenol manufactured by Monsanto
4 between December 1, 1978 and April 1, 1979 contain total
5 tetradioxins in a range from none detected to as high as
6 approximately 500 parts per billion. The latter having 120
7 parts per billion coeluting with 2,3,7,8 TCDD. Assume that
8 this material was manufactured in a process which used
9 caustic, which use was discontinued after April 1, 1979.
10 Assume that an isomer specific analysis was performed in
11 February of 1981 on a sample of 2,4 dichlorophenol showing
12 total tetras of 149 parts per billion and 14 parts per
13 billion coeluting with 2,3,7,8 TCDD. I am sorry, let me
14 repeat that. I said it was an isomer specific analysis so it
15 wouldn't be coeluting. It found 149 parts per billion total
16 tetras and 14 parts per billion of 2,3,7,8.

17 Assume that levels coeluting with the 2,3,7,8
18 standard have been found in 2,4 dichlorophenol as high as 210
19 parts per billion. Assume that the 2,4 dichlorophenol was
20 shipped by tank car and was used in an enclosed system for
21 the production of 2,4-D, a mean product.

22 Based upon your studies of the use of phenoxy
23 herbicides, the levels of dioxins found therein and the
24 toxicological significance of dioxin, do you believe that the

1 dioxin levels as stated above reached the level of
2 toxicological significance to humans in their handling or use
3 of either the 2,4 dichlorophenol or subsequently manufactured
4 2,4-D product?

5 A. Well, this concentration in the dichlorophenol
6 because of the toxicity of the dichlorophenol itself, I would
7 consider it to not have significance. It does not take very
8 much dichlorophenol on the skin or ingested to cause
9 substantial injury and I would consider that this level of
10 contamination in the phenol, per se, would not represent a
11 significant part of the toxicity of the total.

12 Q. Doctor, I want you to further assume -- well, first
13 of all, what about in the subsequently manufactured 2,4-D
14 product?

15 A. In the 2,4-D product, and we are assuming that this
16 is carrying forward in the product at 210 parts per billion,
17 is that correct?

18 Q. Well, would you assume that?

19 A. Not necessarily.

20 Q. And why not?

21 A. Well, there is a reaction that takes place that
22 adds another molecule.

23 MR. CARR: I object here unless this witness is
24 qualified as a chemist of some sort. I think he hasn't and I

1 object to this answer.

2 MR. HEINEMAN: Your Honor, he is clearly
3 qualified. He has a background in studying the phenoxy
4 herbicides and he has had chemical training as testified to
5 on the very first day of his testimony.

6 THE COURT: Do you have anything further you want
7 to say?

8 MR. CARR: I don't recall it but if he has some
9 training that makes him capable of answering that question, I
10 will withdraw it but I don't recall anything, any chemical
11 training.

12 MR. HEINEMAN: Well, my recollection, Your Honor,
13 he testified both when he got his Doctor of Veterinary
14 Medicine and his Masters in Physiology, he had extensive
15 study in the area of chemistry among others, pharmacology,
16 biochemistry, physical chemistry. That is my recollection of
17 his testimony and, of course, his experience with
18 agricultural chemicals.

19 MR. CARR: I have nothing further, Your Honor.

20 THE COURT: Objection is sustained.

21 MR. CARR: Your Honor, I won't object if counsel
22 establishes it in more detail but I certainly don't want to
23 keep this testimony out if this witness, if --

24 THE COURT: If you wish to lay a further

1 foundation, you may. Go ahead under your original
2 hypothetical.

3 Q. Doctor Dost, would you tell the Court and the jury
4 the training that you have had in chemistry in the course of
5 obtaining the degrees that you have, that you have achieved?

6 A. Well, I have had courses in, of course, the kind of
7 elementary chemistry that any college freshman takes.
8 Analytical chemistry, biochemistry, some physical chemistry
9 and organic chemistry.

10 Q. When did you obtain, when did you take these
11 courses?

12 A. Well, I took some of them, of course, at Washington
13 State University in my undergraduate training. I took some
14 of them at Kansas State University. I have also taken
15 courses while I was on the faculty at Washington State
16 University. I also took some courses in chemistry and
17 biochemistry.

18 THE COURT: Objection is sustained.

19 Q. Doctor Dost, I would like you to further assume
20 that 2,4-D, a mean product -- First of all, Doctor, tell me,
21 sir, do you know whether or not in the manufacture of 2,4-D,
22 that 2,4 dichlorophenol is blended with another material in
23 that process?

24 A. 2,4-D is a more complex chemical than the 2,4 DCP.

1 Another molecule is combined with it to make the 2,4-D.

2 Q. What is that other material?

3 A. It is acetic acid.

4 Q. Do you know, sir, what are the proportions in which
5 the acetic acids and the 2,4 dichlorophenol are added
6 together?

7 A. Well, they are mol for mol. In other words, for
8 every molecule of the phenol, there would have to be a
9 molecule of the acetic acid in the ultimate product.

10 Q. Now, sir, that being true, what would that tend to
11 do to the concentration of a contaminant in the 2,4
12 dichlorophenol?

13 A. It would bring it down to some extent. I would
14 judge on the basis of the molecular weights of the two that
15 it would bring it down perhaps by 25 percent.

16 Q. Now, Doctor, I want you to further assume that the
17 2,4-D, a mean product manufactured from the 2,4
18 dichlorophenol product that I mentioned before, has been used
19 in a 9 percent solution as 2,4 dichlorophenoxyacetic acid in
20 a product called Weed-B-Gone and that this product has been
21 applied by homeowners on their lawns at a rate of four
22 teaspoons per gallon. In your opinion, Doctor, based on a
23 reasonable degree of toxicological certainty, would either of
24 the Weed-B-Gone product or the spray solution reach a level

3
1 of toxicological significance to humans as the result of the
2 presence of dioxins mentioned before in the 2,4
3 dichlorophenol product?

4 A. Well, when we are dealing with the 2,4-D as such,
5 at that concentration, it has a marginal tendency for
6 toxicity. If one were to pour it on the skin and not wash it
7 off, there is some possibility of illness. But toxicity is
8 clearly less than we were talking about with concentrated
9 dichlorophenol. At the same time, the concentration of the
10 TCDD has been diminished very sharply and I don't think that
11 it represents a hazard in the solution as it is sold. It is
12 a registered pesticide and certainly when it is further
13 diluted for distribution, four teaspoons per gallon would be
14 about two-thirds of an ounce in 128 ounces. That would
15 dilute it substantially further and when it is spread, when
16 it is used as a herbicide, sprayed on vegetation and
17 inevitably on the ground, the concentration is going to be
18 very low of TCDD and I don't really think that that
19 represents a significant hazard.

20 THE COURT: Is this a good point for a short
21 break?

22 MR. HEINEMAN: Sure, Judge.

23 THE COURT: We will take a short recess at this
24 time and then resume testimony. The admonishments that I

1 have given you earlier will apply during this break also.
2 Court is in recess.

3 COURT RECESSED:

4 (The following proceedings were had in the hearing
5 and presence of the jury)

6 FRANK DOST

7 having resumed the witness stand, being previously sworn,
8 testified further as follows:

9 DIRECT EXAMINATION

10 By

11 MR. KENNETH R. HEINEMAN.

12 Q. Doctor Dost, I would like to ask you another
13 hypothetical question. I would like you to assume, sir,
14 that during a period from August of 1978 until March 1, 1979,
15 Monsanto manufactured orthobenzo-parachlorophenol known as
16 Santophen-1 by a process using caustic. That there were a
17 number of product samples produced during this period which
18 were analyzed for tetradoxins and found to have levels which
19 ranged from 1.5 parts per billion to 56 parts per billion.
20 That these samples were tested coeluting with the 2,3,7,8
21 TCDD and found to have a range of coeluting with 2,3,7,8 TCDD
22 from not detected to seven parts per billion with the second
23 highest level being 2.9 parts per billion.

24 I want you to further assume, sir, that this

1 material was blended into a solution containing anywhere from
2 2.7 percent to 4.5 percent of Santophen-1 and sold as a
3 disinfectant to hospitals and the public for cleaning
4 bathrooms, nurseries, et cetera. You should further assume
5 that the manufacturer suggested a further dilution of the
6 disinfectant into a water solution of one and one quarter
7 ounces of disinfectant per gallon of water. You should
8 assume that the rate might vary slightly based on actual home
9 or hospital use. You should assume that the manufacturer
10 recommended the use of gloves and the water rinsing of the
11 material in the nursery.

12 In your opinion, Doctor Dost, based on a reasonable
13 degree of toxicological certainty, would either the Santophen
14 product, the disinfectant solution or the recommended
15 application reach a level of toxicological significance to
16 humanbeings including infants as a result of the reported
17 levels of dioxins in Santophen product?

18 A. In my opinion, no.

19 Q. And on what basis do you reach that opinion, sir?

20 A. Well, again this is a product that is highly
21 toxic. This particular phenol just and all phenols are. It
22 is very toxic. The concentration at the highest is still
23 relatively low at seven parts per billion and in the
24 solution, assuming the maximum concentration in which it is

1 marketed, the concentration would be much lower and as it is
2 used, the dilution is still another hundred fold lower and
3 the concentration is down at levels in the low parts per
4 trillion and there simply is not enough of the material there
5 to represent a hazard.

6 Q. Doctor Dost, you have previously testified
7 regarding your work with the Environmental Protection Agency
8 pertaining to 2,4,5-T and the levels of 2,3,7,8 TCDD in that
9 product. During the period from 1970 until 1979, did the EPA
10 have a tolerance level of 100 parts per billion of 2,3,7,8
11 TCDD in 2,4,5-T?

12 A. It wasn't precisely a tolerance level. It was, it
13 was a guideline that was adhered to by all producers. It was
14 never incorporated in the law apparently as nearly as I know
15 but it was a guideline that EPA projected and it was followed
16 by all manufacturers and, as a matter of fact, the
17 concentration is steadily diminished but that was the level
18 that was acceptable until 1979.

19 Q. Was 100 parts per billion?

20 A. 100 parts per billion.

21 Q. Doctor, as a herbicide, was 2,4,5-T registered
22 under FIFRA?

23 A. Yes.

24 Q. Pursuant to that registration, was it not within

1 the power of EPA to require the presence of that 2,3,7,8 TCDD
2 to be placed on the label?

3 A. I would presume that they have that power.

4 Q. Isn't that one of the things that FIFRA is about in
5 registering a product? You have to register the label too?

6 A. The label is a legal document that identifies the
7 product and the way it is to be used and the label really
8 reflects among other things the toxicological character of
9 the substance.

10 Q. Doctor Dost, during the period from 1970 to 1979,
11 did the EPA or any other federal agency require the
12 disclosure of the levels of dioxin present in 2,4,5-T on the
13 labels for that product?

14 A. Not to my knowledge. Those levels were never
15 published on the labels.

16 Q. Did the EPA require, to your knowledge, any notice
17 whatsoever of 100 parts per billion of dioxin being present
18 in 2,3,7,8 TCDD to purchasers of the product?

19 A. Of dioxin in 2,4,5-T you mean?

20 Q. In 2,4,5-T.

21 A. I have never seen such notice and so I assume that
22 it wasn't required.

23 Q. Now, this product, Doctor Dost, was sold to and
24 used by farmers, foresters and many other people for blending

1 and direct application for the killing of weeds?

/ 2 A. That is correct.

3 Q. And it was sprayed. What was done with this
4 product?

5 A. It was sprayed either by aircraft or by machines or
6 backpack apparatus, a variety of methods of application but
7 it was sprayed as a liquid.

8 Q. Now, following the withdrawal of the registration
9 of 2,4,5-T, did the EPA ever require a recall of any 2,4,5-T
10 product from the marketplace?

11 A. To my knowledge, never.

12 Q. Did the EPA ever require any cleanup of any of the
13 property upon which the material was sprayed?

14 A. No.

15 Q. Subject to photodegradation or any other breakdown,
16 microbial or otherwise of this material, as far as anyone
17 knows, is the EPA certainly aware of the possibility that
18 materials sprayed on the soil and grounds of farms in 1979
19 might still be beneath the surface of the soil today?

20 A. Well, I am sure they have access to the same
21 information that we all have access to and so I suppose that
22 they would have to make that assumption of that possibility.

23 Q. Doctor Dost, are you aware of any animal
24 experiments performed with contaminated materials from the

1 Sturgeon spill site?

2 A. I am aware that Doctor Osweiler conducted some
3 experiments with water that I believe emerged from the ground
4 after the thaw in that spring.

5 Q. And where did that water come from?

6 A. It is my understanding that it came from a site --

7 MR. CARR: Again I object to the witness. He
8 obviously doesn't have any knowledge of it. Counsel is going
9 to have to give him the facts. The witness wasn't there and
10 he is not Doctor Osweiler. It has to be hearsay.

11 THE COURT: Objection is sustained.

12 Q. Doctor, I would like you to assume that Doctor
13 Osweiler obtained this water from a ditch which was alongside
14 the area where the tank car came to rest. That the water he
15 obtained contained sediment and that he fed this material to
16 goats and guinea pigs and that none of the guinea pigs died
17 as a result of being fed this material. Would you assume
18 that for me, please, sir?

19 A. Yes.

20 Q. Is there any significance to the result of that
21 study in terms of whether or not there is any hazard to the
22 residents of Sturgeon from dioxin at the spill site?

23 MR. CARR: Your Honor, I object to that because the
24 question does not include the fact that the Osweiler water

1 was never found to have dioxin in it. Clearly the evidence
2 shows that. That is an important factor that counsel must
3 include within the aspect of the question to the witness.

4 MR. HEINEMAN: All right. I will be happy to
5 include that.

6 MR. CARR: He never found 2,3,7,8 TCDD in the
7 water.

8 MR. HEINEMAN: That is right.

9 Q. I will ask you to assume this, too, Doctor, which
10 we already did in the previous hypothetical that was read to
11 you. That Doctor Zabik analyzed that water and found 14
12 parts per trillion of tetras but did not find any 2,3,7,8
13 TCDD in the water, all right. Now, based upon that, do those
14 experiments have any significance in your opinion to whether
15 or not there is a hazard to the people of Sturgeon from any
16 dioxin being present at the spill site?

17 A. The fact that water and sediment contained perhaps
18 -- you ask me the question again so that I can give you a
19 direct answer and then I should perhaps explain the reason
20 for my answer.

21 Q. I am trying to remember the question.

22 (The Court Reporter read back the last question.)

23 A. Yes. They have very real significance.

24 Q. And what is that, sir?

1 A. The fact that there was no 2,3,7,8 TCDD found in
2 the water that emerged from the track area.

3 MR. CARR: Your Honor, I object to that. There is
4 nothing in the hypothetical that shows it emerged from the
5 track area.

6 THE COURT: Objection is sustained.

7 MR. HEINEMAN: I think I did say where the water
8 came from, Your Honor.

9 MR. CARR: It came from a ditch but he is saying it
10 emerged from the track. There is a difference coming from
11 the ditch.

12 THE COURT: I think the question just included the
13 track part. I don't think it included anything. I mean the
14 ditch part, not the track.

15 MR. HEINEMAN: Well, Your Honor, I am sorry.

16 Q. Doctor, assume, if you would, that Doctor Osweiler
17 testified that he asked the EPA to take him to the spot where
18 he could get water from the spill site, from the track, and
19 they took him to this ditch and that is where he got the
20 water that he used in the experiment. All right. Now, with
21 that further assumption, sir, continue with your explanation,
22 if you would?

23 A. The water contained no 2,3,7,8 TCDD. It did
24 contain -- what was the phenol level in that water?

1 Q. 15 percent.

2 A. 15 percent. So the water clearly was associated
3 with the phenol. The fact that no TCDD, 2,3,7,8 TCDD was
4 found indicates to me one of two things, possibly a third.
5 That the phenols in water as they have moved left the TCDD,
6 the 2,3,7,8 behind. The concentration of 14 parts per
7 trillion even if it were 2,3,7,8 would imply that there is a
8 vastly lower level than the 45 parts per billion that was
9 originally in the material and even with the 15 percent
10 factor, we have about a thousand fold decrease in
11 concentration.

12 I would suggest that one of three events occurred
13 and possibly a combination of the three. The phenol in water
14 as it moved left the TCDD behind. The 2,3,7,8. The other
15 possibility is that the analytical data on the reserve sample
16 may have been incorrect. That is a possibility because there
17 was other evidence that the levels were lower. Those are two
18 possibilities. The third possibility is that that material
19 in the ditch in the open was, in fact, subject to
20 photodegradation which resulted in the loss of the 2,3,7,8
21 which is more vulnerable to photodegradation because of the
22 positioning of the chlorines than are other isomers.

23 Q. What is the significance, sir, of feeding the
24 material to guinea pigs?

1 A. Guinea pigs --

2 Q. With no effect?

3 A. Well, guinea pigs are very sensitive. Can you tell
4 me what levels were fed those guinea pigs?

5 Q. I can't, as I sit here right now, no. All right.
6 Doctor Dost, I would like you to assume, sir, that there was
7 a 90 day study in which the high dose -- There was a high
8 dose group of guinea pigs, a low dose group of guinea pigs
9 and a control group of guinea pigs. The high dose group
10 started out at a ratio of one part of this sediment water to
11 10 parts of plain water, was then changed to one part of the
12 sediment ditch water to five parts of plain water and then
13 was changed to a one to one ratio of sediment ditch water to
14 regular water. The low dose group was fed one part ditch or
15 sediment water to 10 parts regular water throughout the
16 experiment and the control group got regular water throughout
17 the experiment. There was no decrease in thymus weight and
18 no significant differences in the weights of the three
19 groups.

20 Based upon that information, sir, would you
21 continue with your explanation.

22 MR. CARR: Your Honor, I will object to that
23 question because there is no testimony that it was sediment
24 water. The testimony is that Doctor Osweiler took it off of

1 the top one foot of the water that was in the ditch. It was
2 at least two feet in depth and that, in fact, the sediment
3 that was found on the filter subsequent to the analysis and
4 the analysis by Doctor Zabik was 70 percent phenols and OCP
5 and could not describe, was unable to describe what the 30
6 percent consisted of.

7 MR. HEINEMAN: Your Honor, if I could respond to
8 that. Doctor Osweiler testified that he put the bucket in
9 the water a foot down when he scooped the water out. That he
10 kept, that there was indeed sediment in the water and that
11 everytime he fed the animals, he stirred the water to get the
12 sediment up into the water.

13 THE COURT: That doesn't meet what the objection
14 was. The objection was sustained. You will have to rephrase
15 it.

16 Q. Doctor Dost, would you assume as true what I have
17 just told you in terms of how Doctor Osweiler obtained the
18 water from the ditch and how he stirred it before he fed it
19 to the animals and that he said there was sediment in the
20 water?

21 MR. CARR: Your Honor, I object unless the witness
22 is further given the significant facts as I have indicated;
23 that that sediment that was, whatever was in the water was
24 filtered out and found to be over 70 percent OCP and phenol

1 and the remaining less than 30 percent, the constituents of
2 it was unknown.

3 THE COURT: Objection is sustained.

4 MR. HEINEMAN: It was not filtered out before it
5 was given to the animals.

6 THE COURT: That doesn't make any difference in
7 that. Objection is sustained.

8 Q. I will add that to it. If you want to add that to
9 it, fine. Assume that as well.

10 A. Well, it is an astonishing level of phenols. I am
11 startled that the guinea pigs tolerated. Their drinking
12 total water supply is one to one mix of a solution that is
13 very, very high in phenols which surprises me. However, the
14 fact that they showed no evidence of decreased thymus weight
15 and no evidence of decreased body weight over a three month
16 period in which they were maintained on this water source
17 indicates to me that there would not have been a significant
18 amount of TCDD, 2,3,7,8 TCDD in that material.

19 MR. CARR: Your Honor, the witness has assumed the
20 fact that isn't true. There wasn't any 2,3,7,8 TCDD in the
21 material as so testified by Doctor Zabik clearly under oath.

22 MR. HEINEMAN: Well, Your Honor, clearly this
23 amounts to a bioassay and that is what -- that is certainly
24 what the analytical result was but it certainly is equally

1 well to say that that is what a bioassay showed as well.

2 MR. CARR: Your Honor, the bioassay cannot show that
3 there was 2,3,7,8 TCDD in that water. Nothing happened to
4 the guinea pigs and the only evidence we have is that that
5 water did not contain any 2,3,7,8 TCDD. That is the only
6 evidence in this case.

7 THE COURT: Objection is sustained. I think that
8 was also part of your hypothetical.

9 Q. Doctor Dost, I would like you to assume that Doctor
10 Silbergeld testified that the water flowing into the Kemner
11 pond would contain 30,000 parts per million of OCP giving a
12 TCDD concentration in the pond of 1.4 parts per billion.
13 Would you assume that what you testified to, sir?

14 A. Uh-huh. Yes.

15 Q. Would you agree with that statement?

16 MR. CARR: What page and date, counsel?

17 MR. HEINEMAN: April 16, page 38.

18 Q. Would you agree with that statement, sir?

19 A. Is this referring to in the spring when the weather
20 warmed up?

21 Q. Yes, sir.

22 A. If I understand these figures, they are implying
23 that the concentration of the phenol that moved into the pond
24 was accompanied by the same concentration of TCDD that was

1 supposedly originally in the tank car. I would disagree with
2 that.

3 Q. And why is that, sir?

4 A. Because the material that moved into, that moved
5 away from the site at the time of the thaw, the material that
6 was left after the excavation, the initial excavation in
7 which so much material was hauled away, is going to be
8 modified by the fact that it has been in the soil. The TCDD
9 will bind to the soil. TCDD will not travel with the OCP. I
10 would suggest that this is the same reason that the TCDD did
11 not appear in the water that emerged beside, that was found
12 beside the track.

13 Q. Now, Doctor Dost, you recall Doctor Silbergeld's
14 testimony with respect to TCDD being in the air because of
15 the burning of railroad ties?

16 A. I remember something to that effect.

17 Q. Do you recall her saying beginning at page 48 on
18 April 16th that when she was asked what happens to the TCDD
19 that is on the ties when the ties burned and the ties is
20 reduced to smoke or vapor and she said that unless the fire
21 temperature reaches a very high level which it would not as
22 far as I can tell from the way you have described this as
23 having occurred because it takes very specific conditions and
24 hazardous waste incinerators, the 2,3,7,8 TCDD would not be

1 changed and I would expect it to enter and be moved about in
2 the environment, attached to particulates from the fire, that
3 is soot and burned organic material from the railroad ties.
4 Do you recall reading that when you reviewed her testimony?

5 A. Yes.

6 Q. Would you agree with that statement?

7 A. I would agree in part because the temperature of a
8 fire of that sort certainly is not high enough to degrade
9 TCDD. Probably also not high enough to degrade all of the
10 other organic material, although some of it certainly would
11 be that is involved with the railroad ties. There is a lot
12 of creosote and other material that is used as a
13 preservative. I question whether the TCDD that might be
14 attached to particulate material, unless it had been totally
15 combusted and was completely inorganic in its nature, I think
16 that that TCDD bound in that fashion would probably be
17 subject to breakdown by light.

18 Q. Why is that, sir?

19 A. Well, because the organic, the organic matrix to
20 which it is attached could serve as a hydrogen donor and I
21 would expect, I would expect photodegradation to take place
22 in such a situation.

23 Q. Let's assume, sir, that for the sake of argument
24 that there would be some TCDD on some organic material there

1 that would not be photodegraded. Would that kind of movement
2 of material in the air create any kind of a hazard for the
3 people of the town of Sturgeon?

4 A. I don't believe so.

5 Q. Why not?

6 A. Well, my experience with smoke and the components
7 of smoke and the potential for respiring smoke or any of the
8 material that is contained in it indicates that material of
9 that sort, smoke is distributed throughout an enormous volume
10 of atmosphere. And the relationship between the respiratory
11 volume, even of a person who is working vigorously which an
12 athlete might very well get a respiratory volume up to 40
13 cubic meters per day or a person who is doing very, very
14 heavy work at home, I am concerned about fire fighters. Even
15 that is a turnover of volume compared to the volume of
16 distribution of that very, very small amount of material that
17 might have become associated with the ties because most of it
18 is going to go right on by. There will be some surely that
19 soaks into the tie. The ties are old and cracked but that
20 total amount is really not large compared to the amount, the
21 total amount that was spilled.

22 Q. You are talking about the amount that would be in
23 the tie versus the amount that would go into the ballast?

24 A. That went right on through into the ballast, the

1 soil under the tracks and so on.

2 Q. Now, if you take that tie and you burn it, sir,
3 would that smoke create a hazard for anybody in the town of
4 Sturgeon?

5 A. I don't think so. I don't think so because it is
6 going to move away. The air, the wind velocity, the average
7 wind velocity in that area is on record and it runs in excess
8 of I think seven miles an hour. The volumes of air that are
9 involved are just enormous and the dilution is so large and
10 the material, even if that burning was to continue for a day
11 or two, the dilution is so great that I find it difficult to
12 imagine anyone coming into contact with an amount of TCDD
13 that has any meaningful possibility of significant exposure.

14 Q. Doctor Dost, based upon the facts that I have asked
15 you to assume in these hypothetical questions with respect to
16 the townspeople of Sturgeon, what is your opinion as to
17 whether or not there is any possibility, any practical
18 possibility of there being any hazard to any of the
19 townspeople of Sturgeon from exposure to 2,3,7,8 TCDD as a
20 result of that spill?

21 A. I don't think that there is any practical
22 possibility of a meaningful exposure. I really don't.

23 Q. Let's assume, Doctor Dost, that the spill had not
24 been cleaned up at all. Assume for the sake of argument that

1 nothing had been removed from that spill site. Would there
2 be any hazard to the people of Sturgeon from the TCDD spilled
3 there?

4 A. Not from the TCDD in my opinion.

5 Q. And why do you say that, sir?

6 A. Well, if you wish, we could perhaps go directly
7 back to the CDC document which represented a one part per
8 billion concentration in residential soil as being the level
9 at which concern should be expressed. They also pointed out
10 that higher concentrations would be acceptable in industrial
11 sites where the exposures are less. A railroad right-of-way
12 is really an industrial site. It has very little common
13 traffic. The material went into the ground. The dilution by
14 soil, if you will, brings the concentration I am sure below
15 or down to at least 10 fold. We know from the concentrations
16 of phenols found in that area that the dilution by soil was
17 at least 10 fold. If we were to simply make a division of 45
18 parts per billion by 10 and assume that the soil contained
19 45, four and a half parts per billion of TCDD, it would not
20 fall outside the criteria that are expressed in the Centers
21 for Disease Control document in my view for an action level,
22 if you will. A level at which some action should be taken
23 to, for protection of people in the vicinity.

24 Q. And what is the safety factor built into the CDC

1 assessment?

2 A. It is about a thousand.

3 MR. HEINEMAN: That is all the questions I have of
4 the witness at this time, Your Honor.

5 THE COURT: Mr. Carr.

6 MR. CARR: Yes, Your Honor.

7 CROSS EXAMINATION

8 By

9 MR. REX CARR.

10 Q. Doctor Dost, I would like to start off by asking
11 you a few questions about your background insofar as it
12 concerns your work with TCDD and 2,4,5-T. Actually, you have
13 in the past characterized yourself as something of a quote
14 instant expert in the area of TCDD, have you not, sir?

15 A. I did that at the time that I became involved with
16 the EPA in 1970, yes.

17 Q. And you became an instant expert not based upon any
18 background with TCDD but based upon the fact that you served
19 on an advisory panel for the United States Department of
20 Agriculture in 1970 and '71, isn't that right, sir?

21 A. I served on that panel, yes, sir.

22 Q. Is the answer to my question that is when you
23 became the instant expert? I know you served on that panel.

24 A. That is when I became an expert.

1 Q. And as you characterize yourself, an instant
2 expert?

3 A. That is correct.

4 Q. And you characterize yourself as an instant expert
5 in 1970 under oath, have you not, sir?

6 A. Yes.

7 Q. Now, at that point in time, what you did in 1970,
8 you had no prior experience with TCDD or dioxin, isn't that
9 correct, sir?

10 A. That is correct.

11 Q. And what you did at that time was for this United
12 States Department of Agriculture was to review the literature
13 that was in existence at that time and not doing any research
14 at all, isn't that correct?

15 A. No bench research, that is correct.

16 Q. No research of any sort other than reading the
17 literature?

18 A. That is correct.

19 Q. All right. And it is the same literature in large
20 part that we know that we have read parts of it at least to
21 this jury. You have seen the Monsanto exhibits. You have
22 seen our exhibits and it is the same kind of literature that
23 this jury has already heard about, isn't that correct,
24 Doctor?

1 A. That is correct.

2 Q. And what you did was to read this literature and
3 summarize it and come to a conclusion, the conclusion that
4 you reached, in 1971 as to whether or not 2,4,5-T should
5 continue to be used in the forest land as it had been used up
6 to that time, isn't that correct, sir?

7 A. The concern went beyond forest land.

8 Q. Well, range land, grassland and things of that
9 sort?

10 A. Yes.

11 Q. And what you did -- and by the time you finished
12 with this review of the literature, the EPA had come into
13 existence in 1971 and your actual report that you and others
14 on this panel made as to the use of 2,4,5-T, your actual
15 report went to the EPA which had just then come into
16 existence and taken over this responsibility, isn't that
17 correct, sir?

18 A. Yes.

19 Q. And you made your opinion that you recommended that
20 the company be continued to allowed to use the 2,4,5-T on
21 these various lands, isn't that correct, sir?

22 A. There were specifications, yes.

23 Q. Is the answer to my question yes, you recommended
24 that they be continued to use this 2,4,5-T on the range land,

1 the forest land and the grassland of this country?

2 A. We recommended it be continued but we made
3 recommendations about conditions that should be applied.

4 Q. And what were the conditions that you recommended?

5 A. That the TCDD levels in the product should not be,
6 a new product should not be higher than a tenth of a part per
7 million and a hundred parts per billion and that if existing,
8 if they were lower than a half a part per million could
9 continue to be sold.

10 Q. What you said was use up what you got but in the
11 future, cut the levels down to a hundred parts per billion on
12 new manufactured 2,4,5-T, isn't that correct, sir?

13 A. Essentially, yes.

14 Q. And the EPA at that time in 1971 accepted the
15 recommendation of your panel, didn't they, sir?

16 A. At that time it did not act on it.

17 Q. Well, at some point in time they did act upon it,
18 did they not, sir?

19 A. Apparently they did.

20 Q. And they allowed the continued use. They used the
21 informal guideline, if you will, of the 100 parts per billion
22 and they allowed the continued use of the more heavily
23 contaminated 2,4,5-T, isn't that correct, sir?

24 A. That is correct.

1 Q. Actually in point of fact, they really took no real
2 official action at all at that point in time. It was just
3 kind of an informal thing that they more or less went along
4 with your recommendation, isn't that correct, sir? By you, I
5 don't mean you specifically, I mean your entire panel?

6 A. I don't know the full mechanics but the substance
7 remained in registration.

8 Q. Is the answer to my question yes, that that is what
9 occurred?

10 A. You used the term informal. I don't know whether
11 it was an informal or a formal action.

12 Q. Well, if it was a formal action, you would have
13 known about it, would you not, sir, being an expert in the
14 field?

15 A. I am not sure that I would. It is a government
16 regulatory action. I know that the chemical was returned for
17 registration.

18 Q. You worked in 2,4,5-T for a number of years after
19 you made this recommendation. You would have known if there
20 had been any formal official EPA action taken on your
21 recommendation, wouldn't you, sir?

22 A. I know that EPA returned it to its registration.

23 Q. That isn't my question, Doctor Dost. I know they
24 returned it. My question is, you would have known if there

1 had been any formal action on this recommendation, wouldn't
2 you, sir, in view of the work that you did subsequently?

3 A. I am not sure that I would have known. I am a
4 toxicologist.

5 Q. All right. Well, now, at that point in time, as
6 far as being a toxicologist was concerned, this was the first
7 time you were asked to offer an opinion from a toxicological
8 viewpoint on 2,4,5-T or dioxin and it again was based upon
9 your search of the literature and not any other kind of
10 activity on your part, isn't that correct?

11 A. That is correct.

12 Q. Now, after you made that recommendation and after
13 you became kind of this instant expert, as you have
14 characterized it, you then were called upon to testify in
15 behalf of a number of chemical companies. In behalf of the
16 Department of Forestry or other departments of that sort
17 against actions brought by various citizens' group who were
18 protesting the use of 2,4,5-T, isn't that correct, sir?

19 A. Yes, sir.

20 Q. Yes. As a matter of fact, in 1976 you testified in
21 favor of the chemical companies in Oregon where the citizens'
22 group brought an injunction action against the use of 2,4,5-T
23 in the United States forest, didn't you, sir?

24 A. No, sir.

1 Q. You didn't testify in behalf of the United States
2 Attorney General's Office where the citizens' group was
3 trying to get an injunction against the use of 2,4,5-T?

4 MR. HEINEMAN: Objection, Your Honor. The question
5 was did you testify in favor of chemical companies. He
6 changed the question.

7 THE COURT: Objection is sustained. Could you
8 rephrase that question.

9 Q. Did you not testify, sir, that 2,4,5-T should be
10 allowed, continued to be allowed to be used in Oregon and
11 elsewhere for that matter in the federal court where the
12 citizens' group sought an injunction against the use of
13 2,4,5-T?

14 A. I testified that I thought it was a safe chemical
15 to use.

16 Q. Could you answer the question as I phrased it. Did
17 you testify against the citizens' group and in favor of the
18 continued use of 2,4,5-T?

19 A. Yes.

20 Q. And it was a citizens' group that brought this
21 action in 1976, wasn't it, sir?

22 A. That is correct.

23 Q. Now, again, in 1977 in California, another
24 citizens' group brought another injunction action trying to

1 prevent the use of 2,4,5-T again in the forest at that time,
2 isn't that correct, sir?

3 A. No, sir.

4 Q. That isn't correct, sir?

5 A. No. The chemical was 2,4-D.

6 Q. All right. Against the use of 2,4-D and 2,4,5-T
7 was not involved at that time?

8 A. No, sir.

9 Q. In any event, it was testimony to allow the
10 chemical companies to continue to manufacture and sell 2,4-D
11 and 2,4-D in 1977 and 2,4,5-T in 1976, isn't that correct,
12 sir?

13 A. No, sir.

14 Q. That isn't correct?

15 A. It had nothing to do with the chemical companies.

16 Q. Doctor Dost, the chemical companies make the
17 product, don't they, sir?

18 A. They make the product.

19 Q. And if it is not allowed to be used in the forest
20 land, they can't sell their product in the United States, can
21 they, sir?

22 A. The issues that I was concerned there with are
23 forestry uses.

24 Q. Answer my question, Doctor Dost.

1 A. Forestry is a small use.

2 Q. I am sorry. What you are saying is in this
3 particular action, the chemical that they are trying to
4 prevent the use of this in forests?

5 A. That is correct.

6 Q. And to that extent, the chemical companies could
7 not sell it for use in forests if the citizens' group had
8 prevailed, isn't that correct, sir?

9 A. Not exactly.

10 Q. Well, how is that incorrect?

11 A. Because this was an action against the United
12 States Forest Service. It had no bearing on commercial, on
13 industrial forestry.

14 Q. Well, I see your point, Doctor Dost. What it
15 boiled down to is the chemical companies couldn't sell the
16 product to the United States Government for use upon all the
17 thousands and thousands and thousands of acres of United
18 States forest if this action were correct?

19 A. In that region, that is correct.

20 Q. Now, that would have affected the ability of the
21 chemical company to sell these chemicals to the United States
22 Government, wouldn't it, sir?

23 A. I am sure it would.

24 Q. And to that extent, it was beneficial to the

1 chemical companies, was it not, sir?

2 A. I guess I would assume so.

3 Q. Now, in 1979 or in 1980, an individual brought an
4 action trying to prevent the State of Oregon from spraying
5 2,4-D on a forest, did he not, sir?

6 A. I am not sure I remember which case you are
7 referring to.

8 Q. Well, maybe I can help you remember it. My notes
9 are not that explicit. I made notes from your deposition and
10 they are not that clear.

11 A. If my deposition depended on my memory, why --

12 Q. We would all be in trouble. Doctor Dost, what you
13 did according to your deposition, this was a case brought by
14 a citizens' group against the Bureau of Land Management in
15 the federal court in Portland. You provided a statement and
16 supplement to that witness's statement. You did not appear
17 in court. You did not give a formal deposition. Does that
18 help you?

19 A. That is correct.

20 Q. All right. Now, that was a suit brought by a
21 citizens' group against the California Department of Food.
22 That must be a different one. There was a subsequent
23 injunction action against the State of California, wasn't
24 there, sir?

1 A. I remember such an action at some point.

2 Q. And there was one case where you testified you were
3 asked to, there was an individual who was being prosecuted.
4 He was trying to prevent 2,4,5-T from being used on the
5 forest land. You blocked access to the state spray crew and
6 he was being prosecuted, a jury trial, and the State of
7 Oregon, you came in and testified against that individual and
8 in behalf of the prosecution, did you not, sir?

9 A. I believe the chemical was 2,4-D.

10 Q. Yes.

11 A. I did that, yes.

12 Q. And then you testified involving an injunction
13 action again against the California Department of Food and
14 Agriculture. That is the document that you provided,
15 correct, sir?

16 A. I did provide a document in that action, yes.

17 Q. And then there was in '79 there was a citizens'
18 group in Portland Oregon that brought suit against the Bureau
19 of Land Management to prevent the use of 2,4-D, was there
20 not, sir?

21 A. Yes.

22 Q. And then you testified in that case against the
23 citizens' group, did you not, sir?

24 A. That is correct.

1 Q. And in 1982, and that must be the California action
2 where you provided an affidavit to be used against the
3 citizens' group that was suing the California Department of
4 Agriculture, isn't that correct, to prevent the use of 2,4-D?

5 A. I am very vague about the dates and the times but I
6 remember such actions.

7 Q. All right. Now, in 1978 or '79, the EPA finally
8 did reject your 1971 recommendation and, in fact, suspended
9 the use of 2,4,5-T on forest land, didn't they, sir?

10 A. They suspended the use of 2,4,5-T.

11 Q. Is the answer to my question yes, that they did do
12 that?

13 A. Well, there were two questions, I am sorry.

14 Q. First of all they rejected your 1971
15 recommendation, did they, sir?

16 A. I have no knowledge at all whether they rejected
17 that recommendation.

18 Q. Doctor Dost, you recommended that they be allowed
19 to continue the use of 2,4,5-T in forest land, didn't you,
20 sir?

21 A. Yes.

22 Q. And they did not go along with that in '79 or 78,
23 did they, sir? They said no, we are going to suspend the use
24 of 2,4,5-T in forest land, didn't they, sir?

1 A. I just don't know the reasons why they did that.

2 Q. That was contrary to your recommendation, wasn't
3 it, sir?

4 A. Yes.

5 Q. Now, and since that point in time, you have never
6 been used by the EPA as an expert or in any capacity since
7 that time, have you, sir?

8 A. By EPA?

9 Q. That is correct. By the EPA?

10 A. No.

11 Q. But you have since that time, been -- you have been
12 continued to be used or you are used and have been used by
13 the chemical industry, have you not, sir, and by the forest
14 industry?

15 A. I have served a number of times, yes.

16 Q. As a matter of fact in 1983, and you mentioned that
17 you referred to it in your direct testimony, in 1983 you
18 testified, gave statements in behalf of the Nova Scotia
19 Forest Industries Company, didn't you, sir?

20 A. Yes.

21 Q. And that was a lumber or timber company that wanted
22 to use 2,4 -- wanted to continue to use 2,4,5-T in Canada,
23 isn't that right, sir?

24 A. That is correct.

1 Q. And there was an individual and a citizens' group
2 in Canada just like they did in the United States brought
3 actions against the Nova Scotia Forest Industries, didn't
4 they, sir?

5 A. Yes.

6 Q. And you testified, you came down there and
7 testified on the side of the industry, didn't you, sir?

8 A. Yes.

9 Q. Now, you also were used by the Diamond Alkali
10 Company as an expert witness who was being sued by a lady who
11 became very, very ill after using a herbicide, isn't that
12 correct, sir?

13 A. It was Diamond Shamrock, I believe.

14 Q. Diamond Shamrock, whatever. You did testify in
15 behalf of that company who was being sued by a lady who
16 became very, very ill after using the herbicide, didn't you?

17 A. I did not testify. I provided a witness statement.

18 Q. Provided a deposition, sir?

19 A. Not a deposition. I provided an evaluation of the
20 evidence.

21 Q. Now, you provided an evaluation of the evidence to
22 be used by the chemical company in its defense of its case
23 against this lady who was, as you have described it in the
24 earlier case, very, very ill, isn't that right, sir?

1 A. That is correct.

2 Q. Now, in that case, do you know whether or not the
3 herbicide was made from Monsanto's 2,4 dichlorophenol?

4 A. I have no idea.

5 Q. Well, you do know that that company bought 2,4
6 dichlorophenol from Monsanto, do you not, sir?

7 A. I have been told that.

8 Q. And this herbicide that made this lady -- that she
9 at least said it made her very, very ill, so far as you know
10 was made from Monsanto's 2,4 dichlorophenol, wasn't it, sir?

11 MR. HEINEMAN: Objection, Your Honor, unless we get
12 an identification of what the herbicide is.

13 THE COURT: Objection is overruled.

14 Q. What was the herbicide?

15 A. The herbicide was a substance called MCPA.

16 Q. Do you know whether or not that is made with
17 Monsanto's 2,4 dichlorophenol?

18 A. MCPA is not made with dichlorophenol. It has only
19 one chlorine on it and it as a methyl group on it.

20 Q. And it would have nothing to do with Monsanto
21 then?

22 A. I really don't know. I have no knowledge about the
23 sources of those chemicals.

24 Q. I have no knowledge that Monsanto made any such

1 product and so the answer to -- if this answer to my question
2 so far as you know, Monsanto's chemicals were not involved,
3 isn't that correct, sir?

4 A. Yes.

5 Q. All right. Now, you have also given a deposition
6 or, you couldn't have testified because the case didn't go to
7 trial but you gave an expert deposition and served as a
8 consultant on the side of Monsanto and these other chemical
9 companies, on the side of the chemical companies and against
10 the Vietnam veterans in the Agent Orange case, isn't that
11 correct, sir?

12 A. Yes.

13 Q. And you have also testified for Monsanto and
14 against the workers, employees, in Nitro, West Virginia,
15 haven't you, sir?

16 A. Yes.

17 Q. Now, have you ever, since you became an expert in
18 1970 or '71 on dioxin in the fashion that you have described,
19 have you ever testified in behalf of an individual or a
20 citizens' group involved with dioxin or dioxin contamination?

21 A. Not with those chemicals. With others, though.

22 Q. Have you ever served as a consultant to the EPA
23 except on the occasion when the United States Department of
24 Agriculture started in 1970 and was taken over by the EPA?

1 A. No.

2 Q. Have you ever served as a consultant to any
3 governmental agency, testified before a Congressional
4 committee or done any kind of government work or service
5 since 1979 when the EPA suspended the use of 2,4,5-T?

6 A. Other than natural resource agencies, Bureau of
7 Land Management, forest service, state agencies, no.

8 Q. All of that was before the EPA suspended the use of
9 2,4,5-T, was it not?

10 A. No. That activity continues right to this day.

11 Q. In behalf of whom?

12 A. Behalf of the United States Forest Service, the
13 Bureau of Land Management, State forestry agencies, British
14 Columbia Administry of Forests, the Department of
15 Environmental Quality in Oregon.

16 Q. You are testifying involving 2,4,5-T?

17 A. You asked me if I served as a consultant to those
18 agencies. Did you specify 2,4,5-T?

19 Q. I think you are probably right. I don't believe I
20 did specify 2,4,5-T. You have served since 1979 as a
21 consultant to one of these, one or more of these agencies,
22 sir?

23 A. Yes. The Bonneville Power Administration which is
24 a branch of the United States Department of Energy.

1 Q. Well, what it was in 1982, you served as a
2 consultant on herbicides for the Bonneville Power
3 Administration?

4 A. I still do. I do that on a standing basis as
5 essentially part of my job.

6 Q. Doctor Dost, to talk for a few minutes about your
7 background so that we can get that clear, the expertise that
8 you have acquired in TCDD that we are concerned here with did
9 not come from any scholastic or academic training or courses,
10 did it, sir?

11 A. No.

12 Q. You have a veterinarian degree and by virtue of
13 that fact, you are properly called doctor, are you not, sir?

14 A. Yes.

15 Q. Because you have a DVM, isn't that correct, sir?

16 A. That is correct.

17 Q. But you do not hold a Ph.D.. That is a doctors
18 degree in any scientific field, do you, sir?

19 A. No.

20 Q. You hold what you do hold, the only advanced
21 academic degree that you hold over and above your
22 veterinarian medicine degree is a Masters Degree in
23 veterinary physiology, isn't that correct?

24 A. In physiology, yes.

1 Q. And, of course, you are aware of Doctor Silbergeld
2 whose testimony you read and critiqued, if you will, for the
3 benefit of this court. You are aware that she does hold a
4 Ph.D. and that it is based upon original scientific
5 research. You do know that, don't you, sir?

6 A. Yes.

7 Q. And you do not hold a degree of that nature, do
8 you, sir?

9 A. No, sir.

10 Q. Now, have you ever taken any university taught
11 courses in toxicology?

12 A. Not in toxicology, per se.

13 Q. And have you ever taken any university taught
14 courses in epidemiology?

15 A. No, sir. I have never represented myself as an
16 epidemiologist.

17 Q. Doctor, of necessity, when you testify as a
18 toxicologist, you have to have or do you consider that you
19 have to have knowledge of epidemiological studies in order to
20 determine the toxic consequences of exposure to certain
21 chemicals?

22 A. If one is considering direct effects on humans in
23 using that data, one needs some expertise in epidemiology.

24 Q. And you don't consider that you have any expertise

1 in that field?

2 A. I have general expertise but I don't consider
3 myself an epidemiologist.

4 Q. I didn't ask you that, sir. In order -- because
5 this case is about human health effects on a group of
6 people. Of necessity for you to testify in this case, you
7 have to have some knowledge of epidemiology, must you not,
8 sir?

9 A. I don't think so.

10 Q. You don't think so. Then all of the opinions that
11 you have been giving here have not been based upon the human
12 health studies that are in existence?

13 A. They are based on human health studies, animal
14 studies. The human -- as I see the human, when I read the
15 scientific literature, is one of many species. I don't try
16 to interpret population effects.

17 Q. And nothing, no testimony that you have given here
18 then has been based upon any consideration from an
19 epidemiological viewpoint?

20 A. I don't believe so.

21 Q. All right. And you have, of course, not be taught
22 any such courses in any university, isn't that correct, sir?

23 A. That is correct.

24 Q. Now, have you, yourself, ever taught any

1 toxicological courses at a university?

2 A. Yes, I have.

3 Q. What courses have you taught, Doctor?

4 A. I have taught some special topics courses at Oregon
5 State University in the graduate program in toxicology. We
6 have a training grant at Oregon State University that has
7 been going on for a long time and I have taught students in
8 that. I have taught post doctoral fellows in my laboratory
9 and graduate students in toxicology.

10 Q. And when was that, Doctor?

11 A. Up until 1980. I have not taught formally in the
12 classroom. In fact, I did little formal class room teaching
13 at Oregon State University. I have conducted special topics
14 courses for students in the toxicology program leading them
15 on special assignments.

16 Q. Doctor, according to the CV that I have been given,
17 since 1980 you have been an extension specialist. That means
18 you don't teach courses, you go out in the field?

19 A. That is exactly right.

20 Q. Since 1980. That has been the last five years you
21 haven't taught at all, isn't that correct?

22 A. I have not taught formal courses. I have taught
23 physicians. I have taught foresters, farmers, managers.

24 Q. Well, that is an agricultural extension service

1 that is given to the public in Washington or Oregon, isn't
2 that correct, sir?

3 A. Yes.

4 Q. From '75 and until '80 you served as a professor in
5 agricultural chemistry and veterinarian medicine, didn't you,
6 sir?

7 A. The toxicology program is centered in the
8 Department of Agriculture Chemistry.

9 Q. Well, is there some description in anything that
10 you have given us that will suggest that you taught any
11 toxicological courses, sir?

12 A. No.

13 Q. And the experience that you have had insofar as,
14 well, for instance, neurotoxicology, have you ever done any
15 research other than reading literature in neurotoxicology,
16 for instance?

17 A. We did research on hexachlorophene in which we were
18 very concerned about the anatomy.

19 Q. You are saying we, Doctor Dost. So we won't get
20 confused, you are a part of a group, this area here where
21 there is different people have different assignments?

22 A. My colleague was a biochemist.

23 Q. Your colleague was the biochemist. My question is,
24 have you ever done any research in neurotoxicology?

1 MR. HEINEMAN: You mean alone?

2 Q. My question stands on its own, counsel.

3 A. I suppose it is a matter of interpretation. I have
4 done work that involved neural effects. I won't say that I
5 have directed it specifically to neurotoxicology.

6 Q. Doctor, have you ever done any research other than
7 in the field of, other than reading literature in the field
8 of toxicology that is concerned with TCDD?

9 A. No.

10 Q. Or any other polycyclic hydrocarbon?

11 A. Hexachlorophene. I have collaborated on other
12 projects on insecticides. Chlorinated hydrocarbon
13 insecticide. Hexachlorophene work is, I believe, the only
14 paper that I have published in that area.

15 Q. Doctor, have you ever performed any research on the
16 effect of chemicals or chemical substances on either the
17 porphyrins or the immune system?

18 A. No.

19 Q. Have you ever reviewed any scientific documents,
20 literature, anything of that sort for the EPA excepting the
21 1970 or '71 service that was up, ultimately taken over by the
22 -- well, no, I take that back. I mean reviewed scientific
23 documents for the EPA, period?

24 A. Not for the EPA.

1 Q. All right. And except for the work that you did in
2 '70 and '71 in 2,4,5-T, have you ever reviewed any dioxin
3 articles for the EPA?

4 A. Not for EPA.

5 Q. Have you ever performed any service for the Food
6 and Drug Administration?

7 A. No.

8 Q. Have you ever performed any service for the
9 Department of Health and Welfare?

10 A. I have reviewed a couple of documents for NIOSH
11 which is associated with HEW and I served as a consultant for
12 NIOSH.

13 Q. When was that, Doctor?

14 A. That was back in the mid '70s, I think.

15 Q. It was at the time before the EPA rejected your
16 recommendation, was it not?

17 A. Yes.

18 Q. Doctor, have you ever published any article in a
19 peer reviewed scientific journal or done any original
20 research in the field of toxicology concerned with TCDD?

21 A. No.

22 Q. And organizations, sir, are you a member of the
23 National Academy of Scientists?

24 A. No. Very few people are.

1 Q. Well, you know that Ellen Silbergeld is, don't you?

2 A. I am not sure that she is. I would like --

3 Q. Well, she is, indeed.

4 A. Is she?

5 Q. Yes, she is. You are not, you were not aware of
6 that, Doctor?

7 A. No. I am not aware of that.

8 Q. Are you a member of the American Public Health
9 Association?

10 A. No.

11 Q. You know that Doctor Silbergeld is, don't you?

12 A. If you tell me she is.

13 Q. Well, you read her testimony in this case, did you
14 not, Doctor Dost?

15 A. I don't remember reading that but --

16 Q. Well, my question is, you read her testimony, did
17 you not?

18 A. I read her testimony.

19 Q. I am sorry?

20 A. I read her testimony.

21 Q. And you don't recall reading that she is a member
22 of the American Public Health Association?

23 A. I don't remember reading that. I am not sure that
24 I read every word.

1 Q. Are you a member of the American Association for
2 the Advancement of Science?

3 A. Yes, as a matter of fact.

4 Q. When did you become a member of that association?

5 A. When I paid a subscription price for the magazine.

6 Q. Well, perhaps that is your description of the --
7 there is more to running the American Association for the
8 Advancement of Science than paying a subscription to a
9 magazine, isn't there, sir?

10 A. Membership requires only the payment of a fee.

11 Q. Are you a member of the Society of Neuro Science?

12 A. No.

13 Q. Do you hold membership in the American Society of
14 Neuro Chemistry?

15 A. No.

16 Q. Are you part or hold any membership in the
17 International Brain Research Organization?

18 A. No.

19 Q. Do you hold membership in the Society for
20 Occupational and Environmental Health?

21 A. No.

22 Q. Are you part of the assembly of scientists that is
23 part of the National Institute of Mental Health?

24 A. No.

1 Q. Are you a member of the Science Advisory Board for
2 the EPA?

3 A. No.

4 Q. Are you on any NIOSH committees? Are you on any
5 committee of the Occupational Safety and Health Committee for
6 NIOSH?

7 A. No.

8 Q. In your years of service, have you ever received
9 any awards or honors in science dealing with toxicology?

10 A. Well, I consider that my election as chair of the
11 Gordon Conference at a time when the conference was in danger
12 of being suspended because of problems with its quality and
13 its direction, when I was asked to save the conference, one
14 of the most important conferences in the Profession, I
15 consider that a signal honor.

16 Q. Well, is it considered getting an award or an honor
17 in science or toxicology?

18 A. Chairmanship of a Gordon Conference.

19 Q. I am sure it is, Doctor Dost, as far as you are
20 concerned an honor and there is no question about it but what
21 it is is an honor. It is a compliment to you to be made that
22 appointment but my question is more specific. Have you
23 received something that is called honors, sir, or awards?

24 A. No.

1 Q. Now, you know that all of these things that I have
2 asked about, all of these memberships, all the work for the
3 FDA, working for the Department of Health and Welfare,
4 publication in Nature, the Journal of Science, all of these
5 membership things, all of these things you know from reading
6 the deposition of Doctor Silbergeld that she has received and
7 is a member of these various organizations and have done
8 these various things, don't you, sir?

9 A. Uh-huh.

10 Q. Yes. Now, Doctor, so we can make it clear, you
11 have given us earlier the list of documents that -- of course
12 the jury is not aware of this -- the list of articles and
13 documents upon which you base your opinions, isn't that
14 correct, sir?

15 A. Yes.

16 Q. And none of these documents, none of these articles
17 is based upon any work that you have done but all of them are
18 based upon work that others have done and that you have read
19 about?

20 A. That is correct.

21 Q. All right. Now, Doctor, insofar as your opinion as
22 to various things here, is your opinion that dioxin, that is
23 this 2,3,7,8 TCDD dioxin, is the most toxic synthetic
24 substance of which you have knowledge?

1 A. Yes.

2 Q. It is also your opinion, is it not, that it is the
3 most toxic substance that you have ever encountered?

4 A. I am not sure what you mean by encountered, Mr.
5 Carr.

6 Q. Well, you recall your testimony in the case
7 involving the Nitro, Monsanto employees?

8 A. I don't recall all the details.

9 Q. Well, I am sure you could. Do you recall being
10 asked this question. "Of all the things that man has come up
11 with, this may be the most toxic thing that you have ever
12 encountered?" And your answer to that question was, "It
13 could very well be. Synthetic substances, yes". Now, is
14 there any other toxic substance of which you are aware that
15 you have ever encountered that is more toxic of any sort than
16 2,3,7,8 TCDD?

17 A. Well, you are speaking of encountered and I guess I
18 will assume --

19 Q. Work with, I am sure.

20 A. I have concerned myself about. Certain hexatoxins,
21 for example, which is a very potent biological substance that
22 I have had occasion to be concerned about.

23 Q. Is it more potent than 2,3,7,8 TCDD?

24 A. It may be.

1 Q. On what scale, Doctor?

2 A. Well, I think I can't remember exactly but its
3 lethality, I think, is comparable.

4 Q. Now, insofar as the health effects of this 2,3,7,8
5 dioxin, you have found that it affects the skin, haven't you,
6 sir?

7 A. Yes.

8 Q. You have found, you have concluded that it affects
9 the immune system, have you not, sir?

10 A. At what doses?

11 Q. At whatever doses it may affect. You have found,
12 Doctor, you have concluded that it does affect the immune
13 system, haven't you, sir?

14 A. Yes.

15 Q. You have concluded that it affects the liver,
16 haven't you, sir?

17 A. Yes. At sufficient doses.

18 Q. Doctor, if you don't mind, you have found that it
19 affects the liver, haven't you, sir?

20 A. Yes.

21 Q. You have found that it has some cardiovascular
22 effect, haven't you, sir?

23 A. Some, yes.

24 Q. You have found that it affects the gastrointestinal

1 system, haven't you, sir?

2 A. Yes.

3 Q. You have found that it affects the nervous system,
4 haven't you, sir?

5 A. Yes.

6 Q. You have found that it affects the reproductive
7 system and the endocrine. You found that to be true also,
8 haven't you, sir?

9 A. Yes.

10 Q. You found that to be mutagenic to a limited extent,
11 haven't you, sir?

12 A. I would question whether it is mutagenic.

13 Q. Doctor, do you recall your testimony on page
14 21,210, counsel, this question being asked you. "And you
15 found that to be mutagenic. You found that to be true?" And
16 your answer was, "To a limited extent". Wasn't that your
17 answer at that time to that question in Charleston, West
18 Virginia?

19 A. Yes, and that is my feeling now.

20 Q. Well, it was true then that you found it to be
21 mutagenic to a limited extent. It is true today that it is
22 still mutagenic to a limited extent, isn't it, sir?

23 A. All right.

24 Q. Isn't that correct, sir?

1 A. Yes.

2 Q. And you also found it to be carcinogenic, didn't
3 you, sir?

4 A. Yes.

5 Q. Doctor, all of these things -- and you found, of
6 course, that it is teratogenic, didn't you, sir?

7 A. Yes.

8 Q. And you found that it is a promoter of cancer. You
9 found that, didn't you?

10 A. Carcinogenic effect.

11 Q. Is it a promoter of cancer?

12 A. Yes.

13 Q. And you did find that it affects the nervous system
14 including the peripheral system in humans, didn't you, sir?

15 A. I have heard evidence to that effect.

16 Q. Excuse me. My question is, sir, you found that and
17 you accept that as true, do you not, sir?

18 A. Yes.

19 Q. Now, Doctor Dost, many of these things that you say
20 now that you have found to be true, you have just got through
21 saying is directly contrary to a number of things that you
22 said in direct testimony, isn't it, sir?

23 A. No, sir.

24 Q. Well, did you testify that it was mutagenic in

1 direct testimony?

2 A. I said, if I recall, that there had been some
3 studies which showed a mutagenic effect that had not been
4 when attempted to repeat them, they had not shown up
5 positive. I also mentioned the work --

6 Q. Doctor Dost, that isn't what I am asking.

7 MR. HEINEMAN: Objection. He is interrupting him as
8 he is answering the question.

9 THE COURT: Objection is overruled. It was not
10 responsive.

11 Q. Doctor, you said in response to my question that
12 you found it to be mutagenic to a limited extent. Now, in
13 response to the same question now, you are saying that there
14 is literature that is saying that. Now, my question and the
15 question asked you in Charleston, West Virginia, was
16 specific. You found it to be mutagenic to a limited extent,
17 isn't that correct, sir? Isn't that what the question was
18 and isn't that what your answer was at that time?

19 A. In the reading of the literature, that is what I
20 found then.

21 Q. Yes. And, Doctor, if you found it in the Spring of
22 1985, it is still true in the Fall of 1985, isn't it, sir?

23 A. Yes, and I described it in my testimony.

24 Q. Now, Doctor, you told this jury that it doesn't

1 affect the nervous system but yesterday you have just got
2 through saying a moment ago that you found that it did affect
3 the nervous system, didn't you, sir?

4 A. The comment --

5 MR. HEINEMAN: Objection, Your Honor. Mr. Carr
6 when he asked the question doesn't distinguish between
7 animals and humans. The doctor said very clearly in his
8 direct examination, he distinguished between the test results
9 in humans as opposed to the test results in animals and Mr.
10 Carr doesn't ask him that distinction.

11 MR. CARR: And what is it? That it affects of
12 peripheral nervous system in animals and not in humans? Is
13 that what you are suggesting, counsel?

14 A. I stated --

15 THE COURT: Wait a second. Is that the basis of
16 your objection?

17 MR. HEINEMAN: My objection is to the Court is that
18 you are switching the question on the witness.

19 THE COURT: I don't think that is a switching.
20 Objection is overruled.

21 Q. All my question for the record deals with human
22 health effects and those were the questions asked in Nitro,
23 West Virginia, and those are the questions that I am asking
24 today. You are dealing with human health effects, are you

1 not, sir?

2 A. In my testimony here, I have been speaking to
3 animal effects. The toxicology in animal.

4 Q. And never talking about human effects?

5 A. On rare occasions I spoke.

6 Q. Doctor Dost, are you now saying that we should
7 consider that none of your testimony relates to humans?

8 A. Of course not.

9 Q. You have testified time and time again as to
10 relation to humans, did you not, sir?

11 A. I pointed out the humans' ^{HAVE} ~~half~~ with the same kind
12 of systems that mammals do.

13 Q. And you meant for your testimony to be applicable
14 to humans. You meant for this jury to believe, to take your
15 expertise and tie it in to the humans, here in this case the
16 plaintiffs, that are alleging, claiming certain health
17 effects. You meant that, didn't you, sir?

18 A. In my description of the animal toxicology, that is
19 what I would expect.

20 Q. And the long hypothetical question that was asked
21 you, you understood that the plaintiffs' were humans that
22 were described in that question. You understood that,
23 didn't you, sir?

24 A. Yes.

1 Q. And you have found, have you not, that the human
2 peripheral nervous system is affected by dioxin. You have
3 found that too, have you not, sir?

4 A. My recollection of the information is that in the
5 Nitro workers --

6 Q. Excuse me, Doctor Dost, could you answer that
7 question and then if you want to explain it, please explain
8 it, but you have found that, haven't you, sir?

9 A. Yes, and if I may explain?

10 Q. Yes.

11 A. And I didn't bring this up in my direct testimony
12 in this case because I had forgotten it but in the
13 examination of the workers at Nitro, I did mention one
14 individual who had a nerve biopsy and that individual was
15 found to have some demyelination. The evidence of nervous
16 effects in humans in the Nitro case was reported on
17 interviews with the patient who described aches and pains,
18 tingling of the extremities and such symptoms. I do not know
19 the results of the clinical examinations of those
20 individuals. I do not remember what the clinical
21 examinations showed. On that basis, it can be assumed that
22 those individuals had nervous effects.

23 Q. And, Doctor, you found in addition and you
24 testified in Nitro that you are convinced that it does have

1 the ability to exert a toxic effect on the peripheral nervous
2 system in humans, didn't you, sir?

3 A. I don't remember that. It is not unreasonable.

4 Q. It is a fact. You are convinced, aren't you, sir?

5 A. If that testimony is so, then I will accept that.

6 Q. Notwithstanding that testimony. Just assume that I
7 am making that up. You are convinced, aren't you, sir, that
8 TCDD does have a toxic effect on the human peripheral nervous
9 system, aren't you, sir?

10 A. I am not.

11 Q. You are not. Now, when you were under oath in
12 Nitro, West Virginia, you were convinced, weren't you, sir?

13 A. Yes, and I have given it a great deal --

14 Q. Doctor Dost, if you don't mind.

15 A. Yes.

16 Q. Have you done some new research since the spring of
17 1985 to unconvince you? You are convinced in the Federal
18 court in West Virginia and you are not convinced in the State
19 court in Illinois. What has occurred -- well, let me ask it
20 a different way. Have you done any research since then, sir?

21 A. Only reading.

22 Q. Have you read any new articles since then, sir,
23 that would suggest that there is not an effect upon the
24 peripheral nervous system of humanbeings?

1 A. All --

2 Q. If so, please name the article?

3 A. I have read animal toxicology studies.

4 Q. We are talking about humans.

5 A. I have no evidence in humans, per se.

6 Q. Now, have you read some animal data that would
7 convince you or make you no longer convinced that it has this
8 toxic effect upon the peripheral nervous system in humans?
9 If so, please state the name of the studies?

10 A. I have read -- I have reread a number of papers.
11 The studies on monkeys. I reread all of the pathology
12 studies including Doctor Koceba's work, including the
13 National Toxicology Program work, and I do not see evidence
14 in those studies for effects on animals in the peripheral
15 nervous system.

16 Q. Doctor, you read all of those studies. All of
17 those studies are old. There is none of them new.

18 A. That is correct.

19 Q. They are many, many years old. You knew about them
20 in 1979 and you knew about them in 1980 and you knew about
21 them in '81, '82, '83, '84, '85. None of those things are
22 new. You reread those and you had knowledge of all of those
23 things when you testified in the Federal court, did you not,
24 sir?

1 A. I did.

2 Q. And you reread those articles for the purpose of
3 testifying in this case, didn't you, sir?

4 A. No. Actually not. I have been concerned about
5 neural effects for a long time and I have gone back
6 independently of this case to try to come to some conclusion.

7 Q. But now you are no longer convinced that it has an
8 effect upon the nervous system, is that right, sir?

9 A. In the nervous system of animals.

10 Q. No, of humans. I am talking about humans because
11 you were convinced in the Federal court that it had the
12 effect upon humans. I am talking about humans. I don't give
13 a hoot about the animals, Doctor.

14 A. Well, animals are what I work with.

15 Q. Doctor, I understand that. But my question is
16 specific. Are you now convinced or are you unconvinced that
17 the peripheral nervous system in humans are not affected by
18 TCDD?

19 A. I say -- I think the word that you have used
20 unconvinced is perhaps more appropriate.

21 Q. All right. You were convinced in the spring of '85
22 but you are not convinced today?

23 A. I am no longer certain.

24 Q. And the only thing that has caused you to become

1 from convinced to no longer certain is your rereading of
2 these studies that you have mentioned?

3 A. That is correct.

4 Q. Is that correct, sir?

5 A. That is correct.

6 Q. Now, Doctor, you knew that these men that were
7 exposed in Nitro, West Virginia, you knew they complained of
8 peripheral neuropathy and peripheral neuritis along with the
9 finding that the myelin sheath in a biopsy was damaged and
10 destroyed. You knew those things, didn't you, sir?

11 A. That is correct.

12 Q. You also know that those men today -- how many
13 years later, 26 years later, no more than that, 36 years
14 later are still complaining about the peripheral neuritis and
15 the pains and the aches, aren't they, sir?

16 MR. HEINEMAN: Objection, Your Honor. Are you
17 equating all the Plaintiffs in Nitro with the people that
18 were originally examined by Suskind?

19 MR. CARR: If you don't mind, I will ask this
20 question of the Doctor.

21 THE COURT: Objection is overruled. Please answer
22 the question.

23 Q. You are aware of that, aren't you, sir?

24 A. I am aware that there are complaints. I am also

1 aware of the --

2 Q. Doctor, if you don't mind, my question was, you are
3 aware of the complaints, aren't you, sir?

4 A. I am aware of the complaints.

5 Q. These workers at Nitro, they could be lying about
6 their aches and pains, couldn't they, sir?

7 A. I suppose they could.

8 Q. Or they could be exaggerating, couldn't they, sir?

9 A. Yes.

10 Q. And with humans, however, different from animals,
11 when you look at an animal, unless he is arthritic or have
12 difficulty walking, you really can't tell whether he is or is
13 not having a peripheral neuropathy. By this I don't mean the
14 reflexes but I mean pain in the legs?

15 A. Well, I can tell if an animal is lame. I can't
16 tell if he has minor problems.

17 Q. But if I am walking along right now, if I were a
18 horse or a dog, you couldn't tell whether I have lack of
19 sensation in that foot or pain running down this leg with me
20 walking here?

21 A. Not just observing you, no.

22 Q. But now the humans can tell you that. I mean, they
23 can tell you that they have what you call, what you called, I
24 don't know whether you meant it or not, what you called minor

1 pains. Humans can tell you that they have these so-called
2 minor pains, can't they, sir?

3 A. Yes.

4 Q. Now, Doctor Dost, do you really think that somebody
5 that has had pain down their legs and in their joints for 36
6 years, that you can call that a minor pain?

7 A. No.

8 Q. And it is something, Doctor Dost, that isn't going
9 to show up on any test that you can give that human, isn't
10 that right, sir?

11 A. Well, I am not a clinician but I will assume so.

12 Q. Well, you are enough of a clinician. You studied
13 and have a Masters in physiology, veterinary physiology and I
14 thought you told this jury that the physiology, you take the
15 same courses that the med students do. The same classes and
16 it is much the same. You don't have to be a clinician to
17 know that, do you, sir?

18 A. Well, to be able to do, to make the observations on
19 humans, the laboratory observations that would provide that
20 information, yes.

21 Q. Doctor, I am not aware of any laboratory
22 examination that can tell anybody that I do or do not have
23 pain in my leg.

24 A. I will agree with that, certainly.

1 Q. And, Doctor, you were a doctor. You would have to
2 depend upon the truthfulness of that person, wouldn't you,
3 sir?

4 A. Yes.

5 THE COURT: Mr. Carr, is this a good point to
6 break?

7 MR. CARR: Yes, Your Honor, it is fine.

8 THE COURT: Ladies and gentlemen, we will recess
9 for the day at this time. We will start tomorrow morning at
10 9:30. I would remind you as I do for any overnight break
11 that you are not to read, listen to or watch anything about
12 this case in particular or subject matter in general in any
13 of the media. Thank you for your attention and cooperation.
14 Court is adjourned.

15 COURT ADJOURNED:

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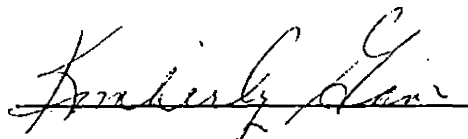
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1 STATE OF ILLINOIS)
2 TWENTIETH JUDICIAL CIRCUIT) SS
3 COUNTY OF ST. CLAIR)
4

5 I, Kimberly Ganz, one of the Official Court Reporters, do
6 hereby certify that the foregoing transcript is a true and
7 correct transcript of the proceedings had in the
8 above-entitled cause.

9 Dated this 13 day of November, 1935.

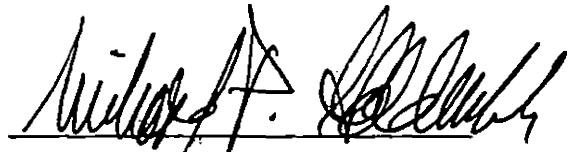
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1 STATE OF ILLINOIS)
2 TWENTIETH JUDICIAL CIRCUIT) SS
3 COUNTY OF ST. CLAIR)
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5 I, RICHARD P. GOLDENHERSH, one of the Judges in and for
6 the Twentieth Judicial Circuit, do hereby certify that the
7 foregoing transcript is a true and correct transcript of the
8 proceedings had in the above-entitled cause.

9 Dated this 13 day of November, 1985.

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13 HON. RICHARD P. GOLDENHERSH
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EXHIBITS

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EXHIBIT NO.

IDENTIFIED

ADMITTED

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1296

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