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November 18, 1993

To: Dr. Dick Clapp

Fm: Carol Van Strum, Investigator

Re: Jan Yung December 28, 1981 memo to William R. Gaffey, with attachments, sent earlier today

This is a privileged and confidential attorney work-product communication with an expert witness in a pending matter. I am writing at the request of Paul Merrell, seeking your advice on documents transmitted earlier today.

Some background on this "missing link" document might be useful, particularly with regard to the musical-chairs shifts in study design for the Nitro studies.

The Zack/Suskind mortality study of 1949 explosion workers with chloracne states (p. 11): "The results of this study will be incorporated with those of a larger study which will include plant workers exposed in the course of 2,4,5-trichlorophenoxyacetic acid production during the period 1948-1969," and (p. 12) "An analysis of the chloracne cases and exposures not associated with this accident but rather with the normal TCP/2,4,5-T production processes will be the subject of a future paper." (2,4,5-T was produced at Nitro from 1948 through 1969.)

The Zack/Suskind study announcing the above future study of TCP production workers with chloracne was published in January, 1980. Ten months later, in October 1980, Monsanto issued a press release proclaiming completion of the second study, identifying it as authored also by Zack and Suskind. (Monsanto medical director George Roush has identified this second study as the one subsequently published as the Zack/Gaffey study.) By the time of the October 1980 press release, however, the study described in the Zack/Suskind study was no longer a study of TCP workers with chloracne, and indeed did not address chloracne at all; instead, it had become a study of mortality in the entire Nitro plant, compared with mortality in a subset of TCP workers identified solely from employment records without any reference to medical condition (i.e., chloracne). The study differed from the study described in the Zack/Suskind study in several significant areas:

1. It did not address chloracne at all;

2. It eliminated from the study altogether all workers who died or ended employment at Nitro from 1948 [first year of 2,4,5-T production at Nitro] to the end of 1954, and from the end of 1977 to the end of 1978 [the cut-off for the Zack/Suskind study], and thus does not Include "plant workers exposed in the course of 2,4,5-trichlorophenoxyacetic acid production during the period 1948 to 1969" as described in the Zack/Suskind study.

3. "Exposure" to TCDD was determined solely by plant employment records (i.e., only workers in the actual TCP process), excluding all others (such as maintenance workers) who had potential exposure and leading to blatant misclassifications.

4. "Exposure" was determined only for a subset of dead workers, not for all TCP process workers (even by the limited employment criteria). [Query from an ignorant layperson: how could the study even calculate a mortality ratio for this subset without a denominator?]

Thus, at some point between publication of the Zack/Suskind study and Monsanto's press release, the study design for the second mortality study had changed radically. (Authorship also changed -- between the time of the press release and presentation of the study at the 1981 Dioxin Symposium, Suskind withdrew his name and Gaffey became co-author.)

For purposes of defending Peter's lawsuit, it has been important to inquire what became of the second chloracne study, and why the design of the second study changed dramatically after publication of the Zack/Suskind study. The Jan Yung memo we sent you earlier today suggests some answers. (Jan Yung was the Monsanto clerk who collected and verified all the data for both the Zack/Suskind and Zack/Gaffey studies.) The memo establishes that Ms. Yung did indeed compile the data for the second chloracne study. Before we take Jan Yung's and Judith Zack's depositions it would be useful to have a rough idea what Yung's data show. Specifically, if her data on the TCP chloracne workers are subjected to the same analysis as the accident worker data in the Zack/Suskind study, what would be the result? And also, if this cohort is added to that of the Zack/Suskind study, what would be the result?

I hope all of this makes some sense to you. Please call if you have any questions!

December 28, 1981

Chloracne cases at the Nitro Plant for years 1950, 1951, 1952, 1953 and 1954

D. S. Fraser - 1670
E. Max Galloway - 1670
V. T. Matteucci - B2SC
E. Tillman, M.D. - G2WE

William R. Gaffey - G2WE

ATTORNEY WORK PRODUCT
ATTORNEY-CLIENT PRIVILEGE

Summary

Following the March 8, 1949 incident at the Nitro Plant, and the cases of chloracne associated with it, additional cases of chloracne began to appear. By 1952 it was evident that the new cases were unrelated to the March 1949 incident, but rather were arising out of exposure in the 2,4,5-T operation. By 1952, there were over 100 claims filed through Workmen's Compensation by employes who had developed a dermatitis or chloracne from exposure in the 2,4,5-T operation.

Plant records prior to 1955 were not routinely retained and therefore an effort was made to identify all 2,4,5-T related claims during the period following the 1949 incident and up to and including 1954. Plant records and Workmen's Compensation records were checked for the years 1950-1954. A total of 570 Workmen's Compensation claims were identified. Of the 570 claims filed during the period 1950 through 1954, a total of 135 white males were identified as having reported dermatitis or chloracne associated with TCDD exposure during normal TCP operations at the plant.

Individual complainants were included in the cohort based on their verified exposure and subsequent chloracne, irrespective of age and number of years employed (Attachment I). The cohort was identified by examining all available plant records, including safety , personnel, and medical records. Workmen's Compensation records were used as an additional source of identifying the cohort and also as a verification of plant records. One individual, a carpenter by the name of Charles Ralph Fowler, filed a claim in 1949, but was not included in the March 8, 1949 TCP accident group. Since he had not been identified previously, he was included in the 1950-1954 group.

Details

The Workmen's Compensation Commission files a record of every claim ever filed in the state of West Virginia, regardless of whether or not payment was awarded. These records are stored in a warehouse in Charleston and are filed by year (approximately 30,000 per year) and are believed to be complete back to 1913. All claims against Monsanto were noted for 1950, 1951, 1952, 1953, and 1954, and copies of the claims were obtained from the Commission. A group of 35 claims were also noted from a group of

claims filed separately under the heading "Skin Diseases". (This group also included several PAB individuals.) All claims with the specific mention of exposure and subsequent dermatitis or chloracne were selected from this group to make up the cohort. It was assumed that any type of injury, no matter how insignificant, would have been reported in the form of a claim to the Compensation Board.

Therefore, these records appear to be the most reliable source of identification and verification of the cohort. Once the cohort was assembled, I contacted Tom Boch and Charlotte Osborne at Nitro as additional sources of verification on questionable exposures.

Results

All of the individuals in the cohort were traced for vital status. Vital status was obtained on all individuals and the breakdown is:

Active Alive	11
Retired Alive	27
Terminated Alive	41
Deceased	56
	135

To date, we have a total of 56 known deceased with death certificates on file. The deceased with cause of death are listed as Attachment V (death certificates attached).

Janet A. Yung

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CHLORACNE CLAIMS FOR

(Identified by Plant Records and

	Name	S S N	Workmen's Comp Claim	Date of Injury	Diagnosis	Race /Sex
1	Amos, Walter E.	232-40-8226	13899-71	10-20-51	Dermatitis	WM
2	Arthur, Charles E.	233-24-3554	13891-192	10-12-51	Dermatitis	WM
3	Arthur Sr., Herman	236-09-2140	14163-29	07-10-52	Dermatitis	WM
4	Arthur, John	232-48-7378	14107-200	05-15-52	Dermatitis	WM
5	Asbury, Elton	232-16-0917	14167-37	07-14-52	2,4,5-T rash	WM
6	Asbury, James F.	232-18-8598	13768-225	06-11-51	Chloracne	WM
7	Asbury, Raymond L.	233-36-3116	13876-182	09-27-51	Dermatitis	WM
8	Bailey, Ervin G.	233-38-5175	14043-178	03-12-52	Dermatitis	WM
9	Bailey, Howard	232-14-5787	Med Records	1951	Chloracne	WM
10	Bailey, Lyal Garland	234-34-2726	14204-23	08-20-52	Dermatitis	WM
11	Bailey, Shelby R.	236-40-5614	14259-75	10-14-52	Dermatitis	WM
12	Bartram, Thomas W.	235-09-0373	Plt Records	03-13-52	Chloracne	WM
13	Beckman, Ervin W.	235-09-0255	14132-88	06-09-52	Dermatitis	WM
14	Boch, Thomas	278-14-1520	14703-20	1952	Dermatitis	WM
15	Brewer, Charles E.	234-22-9849	Med Records	09-29-52	2,4,5-T irri.	WM
16	Burgess, Leland	236-22-8211	13836-72	08-18-51	Dermatitis	WM
17	Casto, Erval	234-26-8710	14216-16	09-01-52	Dermatitis	WM
18	Cavender, Wilford C.	236-24-3261	14154-52	07-01-52	Chloracne	WM
19	Cobb, Ottis B.	236-30-2316	14164-122	07-11-52	Chloracne	WM
20	Cochran, Hansford W.	234-48-8319	Med Records	11-29-51	2,4,5-T rash	WM
21	Collins, John H.	155-22-1379	14227-214	09-12-52	Dermatitis	WM
22	Cook Jr., George K.	232-46-5031	13950-179	12-10-51	Dermatitis	WM
23	Criner, Homer K.	232-12-8440	14642-20	11-06-52	Dermatitis	WM
24	Crossler, Charles	234-48-5923	Med Records	03-05-51	2,4,5-T rash	WM
25	Cunningham, Kenneth C.	233-34-1084	13946-171	12-06-51	Dermatitis	WM
26	Cyrus, Fred W.	232-40-0891	14045-166	03-14-52	Dermatitis	WM
27	Davis, Howard D.	236-26-2333	13583-109	12-08-50	Dermatitis	WM
28	Davis, Park B.	233-16-3596	14089-17	04-27-52	Chloracne	WM
29	Davis, William	232-34-2710	14444-89	04-17-53	Dermatitis	WM
30	Dent, Thomas A.	235-09-0268	13859-192	09-10-51	2,4,5-T rash	WM
31	Dickerson, Isaiah	232-12-8446	13485-118	09-01-50	Dermatitis	WM
32	Eggleston, George F.	234-48-5170	14399-173	08-11-53	Chloracne	WM
33	Ennis Jr., Andrew A.	232-32-1908	13902-187	10-23-51	Dermatitis	WM
34	Farley Jr., Charles E.	232-32-3686	13509-171	09-25-50	Dermatitis	WM
35	Ferrell, George L.	280-26-8400	14102-49	05-10-52	Chloracne	WM
36	Fisher, Floyd R.	234-20-2488	Plt Records	02-23-50	Dermatitis	WM

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PERIOD 1950 - 1954

Workmen's Compensation Claims)

M/S	Date of Birth	Date of Hire	Job Title	Employee Status	Vital Status
M	05-04-23	07-31-50	Helper	Active	Alive
M	04-17-21	08-15-50	Helper	Retired	Alive
M	09-29-09	04-02-45	Helper	Retired	Alive
M	06-18-29	03-29-50	Helper	Active	Alive
M	01-10-14	10-23-50	Lab (Yard)	Term	Alive SS
M	11-25-17	10-28-46	Helper	Dec'd	Dec'd 1972
M	07-23-24	07-17-46	Helper	Active	Alive
M	01-28-26	01-19-49	Operator	Dec'd	Dec'd 1974
M	02-27-98	02-07-45		Retired	Dec'd 1967
M	08-03-23	08-08-50	Helper	Term	Alive SS
M	01-31-29	07-10-50	Helper	Term	Alive
S	11-26-96	07-05-24	Chemist	Dec'd	Dec'd 1978
S	05-08-08	05-23-33	Foreman	Retired T&PD	Dec'd 1976
M	08-06-15	06-25-51	Supv.	Retired	Alive
M	09-10-21	01-21-41	Insulator	Term	Dec'd 1957
M/S	01-31-24	09-11-50	Helper	Dec'd	Dec'd 1978
M	12-13-19	12-19-46	Helper	Dec'd	Dec'd 1967
M	06-23-23	02-01-46	Operator	Retired	Alive
M	06-01-25	04-12-49	Helper	Dec'd	Dec'd 1976
S	10-23-30	05-10-51	Yard	Term	Alive
M	07-21-29	07-23-51	Chemist	Term	Alive
M	05-02-31	07-17-51	Lab (Yard)	Term	Alive
M	06-13-14	07-12-43	Pipefitter	Retired	Alive
S	09-05-31	09-01-50	Helper	Term	Alive SS
M/S	10-20-25	06-02-47	Helper	Active	Alive
M	06-20-30	07-05-50	Helper	Term	Alive VA
M	03-02-23	11-21-46	Operator	Active	Alive
M	10-10-02	08-16-43	Operator	Retired	Dec'd 1971
M	10-22-28	04-17-53	Helper	Retired	Dec'd 1979
M	01-17-04	02-14-35	Operator	Term	Dec'd 1957
M	10-10-06	01-03-44	Yard	Retired	Alive
M	05-31-31	06-06-50	Helper	Active	Alive
M	09-30-26	07-05-50	Helper	Term	Alive SS
M	04-02-26	10-24-46	Truck Dr	Retired	Alive
M	04-29-25	08-08-50	Helper	Term	Alive SS
M	06-29-20	02-02-49	Helper	Dec'd	Dec'd 1975

	Name	S S N	Workmen's Comp Claim	Date of Injury	Diagnosis	Race /Sex	H/S	Date of Birth	Date of Hire	Job Title	Employee Status	Vital Status
37	Forbes Sr., Willard M.	236-09-2189	13890-210	10-11-51	Dermatitis	Wh	H	12-12-09	05-03-43	Operator	Retired	Alive
	"	"	14939-101	06-25-54	Rash							
38	Fowler, Charles R.	226-03-6107	13014-225	05-18-49	Chem rash	Wh	H/S	02-15-15	04-21-41	Carpenter	Retired	Dec'd 1973
39	Galloway, Howard Max	236-30-4729	14052-178	03-21-52	Chloracne	Wh	S	02-28-24	09-04-51	Supv.	Active	Alive
40	Garton, Dewey T.	233-22-5388	13365-94	05-04-50	Dermatitis	Wh	H	06-03-97	03-02-41	Unloader	Retired	Dec'd 1965
41	Goodall, John F.	232-50-4784	Med Records	08-06-52	Rash	Wh	H	10-12-30	07-24-50	Helper	Term	Alive 88
42	Grant, Harold	234-34-7430	14027-134	02-25-52	Dermatitis	Wh	H	04-03-26	01-12-49	Helper	Retired	Alive
43	Grant, Hansel C.	233-28-9811	13899-70	10-20-51	Dermatitis	Wh	H	10-08-20	11-26-45	Operator	Retired	Alive
44	Hailey, Jesse W.	235-09-0286	Med Records	1952	Chloracne	Wh	H/S	01-05-09	02-03-27	Pipefitter	Retired	Dec'd 1978
45	Harper, Emory	235-03-3934	14701-131	12-24-52	Dermatitis	Wh	H	10-28-13	12-29-44	Operator	Retired	Alive
46	Harris, Earl E.	233-22-9240	13496-100	09-12-50	Dermatitis	Wh	H	08-25-23	04-01-46	Operator	Term	Dec'd 1972
47	Harris, Elmer	235-09-0294	14087-122	04-25-52	Dermatitis	Wh	S	03-12-07	10-10-27	Foreman	Term	Dec'd 1956
48	Harris, George	236-18-1143	14091-187	04-29-52	Dermatitis	Wh	H	07-27-20	01-11-45	Operator	Retired T&PD	Dec'd 1980
49	Harrison, Robert E.	264-40-9012	13941-91	12- -51	Dermatitis	Wh	H	11-28-31	07-19-50	Helper	Active	Alive
50	Hawley, Jasper M.	236-07-6057	14075-14	04-13-52	Chloracne	Wh	H	05-21-09	07-09-33	Pipefitter	Retired	Alive
51	Higginbotham, Clarence	232-40-0884	14007-134	02-05-52	2,4,5-T rash	Wh	H	01-02-30	04-26-49	Helper	Term	Dec'd 1958
52	Higginbotham, George P.	232-12-8883	Plt Records	06-17-52	2,4,5-T rash	Wh	H	11-28-10	02-26-45	Operator	Dec'd	Dec'd 1958
53	Hill, Howard B.	236-24-0895	14107-24	10/50 5/52	Dermatitis	Wh	H	11-28-22	07-26-48	Helper	Active	Alive
54	Hill, Willard B.	234-28-8326	14009-191	02-07-52	Dermatitis	Wh	H	03-12-20	12-20-44	Helper	Term	Alive 88
55	Hoffman, Basil G.	236-26-3311	14384-51	02-16-53	Dermatitis	Wh	H	02-16-25	12-19-46	Helper	Retired	Dec'd 1973
56	Howell, Bernard E.	232-14-5868	13964-164	12-06-51	Dermatitis	Wh	H	08-18-09	12-18-50	Helper	Term	Alive
57	Hundley, Woodrow	285-09-0222	14168-140	07-15-52	Chloracne	Wh	H	08-22-12	09-11-50	Helper	Term	Alive 88
	"	"	14447-191	08-15-53								
58	Johnson, Roy S.	236-09-3339	Med Records	10-08-52	Rash	Wh	H	04-03-07	01-06-47		Dec'd	Dec'd 1955
59	Jones, Byron L.	236-40-1268	13904-184	10-25-51	Dermatitis	Wh	H	01-15-31	04-17-50	Truck Dr	Term	Alive 88
	"	"	14148-122	06-25-52	Dermatitis	Wh	H		"	"	"	"
60	Lamb, Leonard Wayne	236-40-4057	14226-122	09-11-52	Chloracne	Wh	H	12-10-27	04-10-47	Helper	Dec'd	Dec'd 1966
61	Legg, Zane	236-24-2478	14059-164	03-28-52	2,4,5-T rash	Wh	H	03-16-23	04-29-46	Operator	Dec'd	Dec'd 1958
62	Lett, Chester B.	232-12-8888	13885-79	10-06-51	Dermatitis	Wh	H	11-12-10	10-10-45	Operator		Dec'd 1980
63	Lett, Robert	233-10-9624	13446-177	07-24-50	Chloracne	Wh	H	11-13-13	03-22-41	Insulator	Retired	Alive
64	Lloyd, C. B.	232-14-4119	14343-42	01-06-53	Chloracne	Wh	H	07-15-17	06-30-41	Operator	Retired	Dec'd 1978
65	Mallett, John B.	236-38-4463	13798-206	07-11-51	Dermatitis	Wh	H	09-24-22	04-20-44	Pipefitter	Retired	Alive
66	Martin, Hollis H.	236-24-4028	13820-218	08-02-51	Dermatitis	Wh	H	06-19-20	04-24-46	Operator	Active	Alive
67	Martin, Paul E.	233-38-4775	13713-186	04-17-51	Dermatitis	Wh	H	09-08-26	04-02-47	Helper	Term	Dec'd 1957
68	McClanahan Jr., Anthony	232-34-4109	13295-68	02-23-50	Chloracne	Wh	H	08-03-26	01-06-47	Laborer	Term	Alive
69	McClanahan, Denver	232-24-6845	14185-165	09-04-52	2,4,5-T rash	Wh	H	03-03-22	12-01-47	Helper	Term	Dec'd 1954
70	McClanahan, Elmer A.	236-18-0129	14087-145	04-25-52	Dermatitis	Wh	H	05-24-22	04-09-51	Helper	Term	Alive
71	McClanahan, Harold C.	236-38-4555	13719-182	04-23-51	Dermatitis	Wh	H	01-06-28	12-01-47	Helper	Term	Alive
72	McClanahan, J. Harold	236-42-6451	14017-204	02-15-52	Dermatitis	Wh	H		07-10-50	Helper	Term	Alive

	Name	S S N	Workmen's Comp Claim	Date of Injury	Diagnosis	Race /Sex	H/S	Date of Birth	Date of Hire	Job Title	Employee Status	Vital Status
73	McClanahan, Millard R.	234-22-9974	13968-158	12-28-51	Dermatitis	WM	H	01-11-21	02-02-49	Helper	Term	Dec'd 1966
74	McClanahan, William R.	236-24-7509	14070-85	04-08-52	Chloracne	WM	H	11-27-21	04-16-51	Helper	Term	Alive
75	McCoy, Hansford L.	234-34-1049	14023-166	02-21-52	Dermatitis	WM	H		08-15-51	Welder	Term	Alive 88
76	McCoy, Harold	236-40-6398	14336-172	12- -52	Dermatitis	WM	H		05-15-50	Helper	Term	Alive
77	McCoy, John E.	234-34-1048	13491-177	09-07-50	Chloracne	WM	H	04-26-24	05-12-47	Helper	Retired	Alive
78	McGrew, James E.	236-09-7723	13789-79	07-02-51	Chloracne	WM	H	07-24-12	03-29-45	Electrician	Term	Alive
79	McLaughlin, Ralph L.	235-05-0258	14260-219	10- 52	Rash	WM	H	08-20-19	10-10-50	Helper	Term	Alive
80	Meadows, Basil	234-26-6868	14822-114	04-03-54	Dermatitis	WM		09-23-12	02-17-45	Helper		Dec'd 1957
81	Meadows, Devey	236-24-1315	14136-31	06-10-52	2,4,5-T	WM	H	12-25-24	01-22-45	Pipefitter	Retired	Alive
82	Melton, Woodrow W.	235-09-0322	14449-101	04-22-53	Dermatitis	WM	H/S	11-26-13	05-13-33	Repairman	Dec'd	Dec'd 1971
83	Miller, Roy H.	236-09-2257	14308-57	12-02-52	Dermatitis	WM	H/S	05-24-15	04-24-37	Foreman	Term	Dec'd 1961
84	Miller, Thomas	236-09-2263	14982-142	10-07-54	Chloracne	WM	H	07-22-15	03-25-46	Operator	Dec'd	Dec'd 1957
85	Newcomer, Harold L.	233-12-3560	14318-31	12-12-51	Dermatitis	WM	H	06-06-19	07-31-50	Helper	Term	Dec'd 1974
86	Null, Emmett	233-18-1788	14434-57	04-07-53	Dermatitis	WM		10-19-17	07-10-50	Operator	Retired	Alive
87	O'Dell, Roy	235-05-5530	14589-4	08-27-53	Dermatitis	WM		08-20-16	04-24-46	Operator	Retired	Alive
88	Painter, Arnie	233-28-0533	14541-215	07-23-53	Chloracne	WM		12-11-20	02-23-45	Helper	Term	Alive 88
89	Painter, Gay	236-16-0010	13856-196	09-07-51	Dermatitis	WM	H	02-18-18	07-24-50	Laborer	Term	Dec'd 1973
90	Parkins, Shelly	232-18-8034	14148-74	06-25-52	Dermatitis	WM	H	03-08-99	07-12-43		Term	Dec'd 1967
91	Payne, Donald	234-34-4080	13932-27	11-22-51	Rash	WM	H	08-27-21	01-12-49	Helper	Active	Alive
92	Payne, Kendall E.	233-44-2100	13803-244	07-16-51	Dermatitis	WM	H	07-26-29	04-16-51	Helper	Term	Alive SS
93	Persinger, Sherman	236-07-5795	13991-26	01-20-52	Dermatitis	WM	H	12-24-08	10-23-50	Helper	Term	Alive
94	Phillips, Letcher L.(2)	234-16-0847	14125-166	06-24-52	Rash	WM	H	11-24-10	03-02-44	Pipefitter	Retired	Dec'd 1978
95	Ranson, R. B.	235-01-3846	14589-5	07-01-53	Chloracne	WM	H	01-03-01	03-21-44	Helper		Dec'd 1963
96	Rhoads, Charles M.	236-22-6194	14308-58	12-02-52	Dermatitis	WM	H	12-22-21	10-16-50	Helper	Term	Dec'd 1977
97	Richardson, Roy M.	236-09-2309	14046-62	03-15-52	Chloracne	WM	H	08-30-04	07-12-43	Pipefitter	T&PD	Dec'd 1964
98	Roberts, Guy L.	232-16-9637	13429-90	08-02-50	Chloracne	WM	H	09-06-97	02-22-37	Pipefitter	Dec'd	Dec'd 1974
99	Saxton, William E.	235-44-7659	14001-176	01-30-52	Dermatitis	WM	H	08-20-22	09-25-50	Helper	Term	Alive SS
100	Sayre, Pearly	233-36-9532	13838-77	08-20-51	Irritation	WM	H	06-16-26	04-10-47	Helper	Term	Alive
101	Sayre, Reuben D.	235-05-3654	14078-163	04-16-52	Dermatitis	WM	H	08-23-15	12-21-42	Operator	Retired	Alive
102	Scherer, Alfred	236-42-3151	13454-107	08-01-50	Rash	WM	H	11-16-28	06-02-50	Helper	Term	Alive
103	Selbe, Raymond	235-16-2017	14003-168	02- -52	Rash	WM	H		10-23-50	Helper	Term	Alive
104	Shaffer, Willard	235-03-7911	13935-20	11-25-51	Chloracne	WM		06-29-16	02-10-41	Operator	Term	Dec'd 1971
105	Shank, Charles E.	236-09-2316	13655-26	02-18-51	Dermatitis	WM	H	12-28-11	02-18-51	Helper	Term	Alive
106	Shank, Lawrence L. "Roy"	235-09-0342	14349-208	same as 12943-200 on Nitro Accident List -							Retired	Dec'd 1972
107	Sigman, Roy A.	233-16-5759	13890-211	10-11-51	Dermatitis	WM	H	09-15-15	10-23-50	Helper	Term	Alive
108	Simmons, William H.	234-34-2228	13874-198	09-25-51	Dermatitis	WM	H	06-26-25	02-05-44	Operator	Term	Dec'd 1969
109	Smith, William H.	232-44-6136	14168-15	07-15-52	Dermatitis	WM	H	10-01-18	07-24-52	Helper	Term	Alive 88
110	Starcher, Cletus	234-28-9902	13823-25	08-05-51	Rash	WM	H	08-18-18	04-09-51	Helper	Term	Alive
111	Steele, Kermit	235-03-9659	14759-1	02-26-54	Chloracne	WM		08-08-18	10-11-45	Helper	Retired	Alive

	Name	S S N	Workmen's Comp Claim	Date of Injury	Diagnosis	Race /Sex	H/S	Date of Birth	Date of Hire	Job Title	Employee Status	Vital Status
112	Stover, Donald B.	235-05-7576	13665-190	02-28-51	2,4,5-T	WM	H	10-26-10	07-24-50	Helper	Retired	Alive
113	Stover, Enoch	233-10-8632	13537-102	10-23-50	Dermatitis	WM	H	10-09-99	05-10-43	Painter	Retired	Dec'd 1973
114	Stutler, Robert	236-36-7618	13854-192	09-01-51	Chloracne	WM	H	03-25-28	03-27-47	Helper	Term	Alive
115	Swann, Dillard C.	234-28-8117	13978-83	01-07-22	Dermatitis	WM	H	06-11-52		Helper	Term	Dec'd 1969
116	Terry, Willard	293-12-7663	14089-16	04-27-52	Dermatitis	WM	H	07-20-20	04-09-51	Helper	Retired	Dec'd 1980
117	Thomas, Hershel	232-14-9274	13978-92	01-07-52	Chloracne	WM	H	01-01-14	10-30-43		Retired	Alive
118	Tucker, Russell B.	234-24-0204	13631-136	01-25-51	Dermatitis	WM	H	04-26-22	11-12-45	Janitor	Term	Dec'd 1973
119	Vineyard, Hoy M.	236-30-7959	13927-89	11-17-51	Dermatitis	WM	H	09-16-22	10-10-50	Helper	Term	Alive
120	Vintroux, James A.	234-28-8198	14295-123	11-19-52	Rash	WM	H	11-01-14	07-21-45	Operator	Dec'd	Dec'd 1957
121	Waugh, Lawrence M.	235-09-8971	14003-169	02-01-52	Dermatitis	WM	H	07-15-19	09-11-50	Helper	Dec'd	Dec'd 1979
122	Whittington, James E.	718-10-0875	14014-176	02-11-52	Chloracne	WM	H/S	03-27-20	03-25-46	Helper	Retired	Alive
123	Williams, Billy	236-42-5130	14013-159	02-11-52	Dermatitis	WM	H	02-26-30	07-31-51	Laborer	Term	Alive
124	Williams, Earl	236-09-2363	14286-159	11-10-52	Chloracne	WM	H	06-29-19	08-16-43	Pipefitter	Retired	Alive
125	Williams, William L.	232-18-7762	14003-167	02-02-52	Chloracne	WM	H	09-07-15	06-06-50	Helper	Retired	Alive
126	Williamson, Kenton L.	234-28-4337	14045-167	03-14-52	Chloracne	WM	H	04-12-13	03-03-41	Machinist	Retired	Alive
127	Withrow, Corbett	233-36-4120	14611-1	09-22-53	Chloracne	WM	H	02-24-25	01-31-45	Trk Driver	Term	Dec'd 1974
128	Wolfe, Leslie	235-01-4008	14332-80	on Nitro accident list								Dec'd
129	Workman, James L.	232-16-0238	Pit Records	02-04-51	2,4,5-T rash	WM	H	01-23-93	02-28-44	Yard	Retired	Dec'd 1979
130	Workman, John L.	233-32-4578	14006-200	02-04-52	Dermatitis	WM	H	09-26-20	04-11-50	Helper	Term	Dec'd 1971
131	Workman, William W.	236-40-6230	13989-187	01-18-52	Dermatitis	WM	H		07-10-50	Helper	Term	Alive
132	Wright, James R.	235-09-0249	14307-65	12-01-52	Dermatitis	WM	H	09-01-05		Shift Rep	Retired	Alive
133	Wright, Lowell W.	234-48-5234	13874-81	09-25-51	Chloracne	WM	H	12-14-31	05-18-50	Helper	Term	Alive
134	Young, Henry O.	233-10-9644	13761-202	06-04-51	Dermatitis	WM	H	12-05-99	06-12-45		Term	Dec'd 1969
135	Young, James H.	233-36-3690	13914-19	11-04-51	Dermatitis	WM	H	06-18-26	04-16-51	Helper	Term	Alive

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(86)

Chloracne Cases 1950-1954 Deceased

	Name	Date of Death	Age	Cause of Death
1.	Asbury, James F.	08-12-72	54	1621 I. Carcinoma left lung with metastasis.
2.	Bailey, Ervin G.	02-16-74	48	4109 I. Acute coronary occlu. and myo. infarct. ASHD II. Hia. hernia.
3.	Bailey, Howard	12-24-67	69	4124 I. Cerebral vasc. accident. ACVD II. CHF
4.	Bartrum, Thomas W.	10-19-78	81	4369 I. Cerebrovascular stroke. Generalized arteriosclerosis. II. Asp. pneumonia. Heart failure.
5.	Beckman, Erwin W.	12-10-76 PAB	68	1880 I. Cancer of Bladder.
6.	Bewer, Charles E.	02-26-57	35	4109 I. Heart attack.
7.	Burgess, Leland	02-26-78 PAB	54	1880 I. Pulmonary embolism, massive right. Stroke post op. Trans. carcinoma, urinary bladder. II. Status post op. partial cystectomy.
8.	Casto, Erval	11-13-67	47	4109 I. Acute myo infarct.
9.	Cobb, Oatis B.	04-25-76	50	8600 I. Apparent cardio-pulmonary failure. II. Apparent chronic alcoholic poisoning.
0.	Davis, Park B.	11-26-71 PAB	69	4109 I. Myo infarct. ASCVD
1.	Davis, William	04-24-79	50	4109 I. Probable recurrent acute myocardial infarction. ASHD.
2.	Dent, Thomas A.	03-03-57	53	1579 I. Carcinoma liver and pancreas.

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Note: Death certificates attached.

Chloracne Cases 1950-1954 Deceased

Name	Date of Death	Age	Cause of Death
13. Fisher, Floyd R.	08-03-75	55	4109 I. Acute myo infarct. II. AHD
14. Fowler, Charles R.	05-28-73 PAB	58	4109 I. Recurrent acute myo infarct. II. ASHD.
15. Garton, Dewey T.	02-11-65 PAB	67	1880 I. Carcinomatosis. Carcinoma urinary bladder.
16. Halley, Jesse W.	01-17-78 PAB	69	4109 I. Myo infarct. ASHD.
17. Harris, Earl E.	12-08-72 PAB	49	1719 I. Pulmonary embolus. Generalized liposarcoma.
18. Harris, Elmer Lee	10-26-56 PAB	49	4109 I. Coronary thrombosis.
19. Harris, George L.	07-04-80	59	5193 I. Cardio respiratory arrest. Chronic obstructive pulmonary disease and respiratory failure chronic. Corpulmonale. II. Acute bronchitis.
20. Higginbotham, Clarence O.	07-11-58	28	9550 I. Shotgun wound.
21. Higginbotham, George P.	09-20-58	47	4123 I. Coronary insufficiency. Arteriosclerosis, coronary.
22. Hoffman, Basil G.	05-01-73	48	4109 I. Acute myocardial infarction. ASHD.
23. Johnson, Roy S.	07-30-55	48	8199 I. Crushing injury to chest (auto accident).
24. Lamb, Leonard W.	04-06-66 PAB	38	5820 I. Urema. Chronic glomerular nephritis.
25. Legg, Zane G.	07-12-58 PAB	35	8199 I. Fractured skull (auto accident).
26. Lett, Chester B.	01-25-80	69	1621 I. Terminal pneumonia. Carcinoma of lung with severe dysplagia.

Note: Death certificates attached.

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Chloracne Cases 1950-1954 Deceased

Name	Date of Death	Age	Cause of Death
27. Lloyd, Charles B.	04-23-78	60	1621 I. Metastatic carcinoma of lung.
28. Martin, Paul E.	04-27-57	30	9240 I. Terminal pneumonia. Chemical and thermal burns.
29. McClanahan, Denver E.	06-30-54	31	8300 I. Drowning (accident).
30. McClanahan, Millard R.	02-18-66 PAB	45	8109 I. Cerebral contusion (train struck car).
31. Meadows, Basil B.	08-16-57	44	9109 I. Accidental drowning.
32. Melton, Woodrow W.	03-31-71 PAB	57	4109 I. Acute myo infarct. II. Arteriosclerosis, myo infarct., diabetes mellitus.
33. Miller, Roy H.	10-29-61	46	4109 I. Coronary occlusion.
34. Miller, Thomas H.	04-20-57	41	8990 I. 2nd and 3rd degree burns of 70% of body.
35. Newcomer, Harold	04-23-74	54	4109 I. Myo infarct. ASCVD.
36. Painter, Gay F.	02-09-73	54	4109 I. Posterior myo infarct. Coronary artery disease. II. Rt. ureteral calculus.
37. Parkins, Shelly L.	09-03-67	68	4123 I. Acute pulmonary edema. ASHD, CHF, AI. II. Uremia.
38. Phillips, Letcher L.	03-01-78	67	4109 I. Coronary occlusion.
39. Ranson, Robert B.	12-31-63	62	4109 I. Acute myocardial infarction. II. Diabetes mellitus.
40. Rhoads, Charles M.	05-27-77 PAB	55	4109 I. Acute myo infarct. AHD. II. Myo infarct, old, two.

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Note: Death certificates attached.

Chloracne Cases 1950-1954 Deceased

Name	Date of Death	Age	Cause of Death
41. Richardson, Roy M.	08-17-64	59	4109 I. Myo infarct. AED.
42. Roberts, Guy L.	04-25-74	77	4123 I. Ventricular fibrillation. AED. II. Diabetes with metabolic acidosis.
43. Shaffer, Willard L.	01-22-71	54	4109 I. Acute myo infarct. Previous myo infarct.
44. Shank, Lawrence	on Group I list.		4109 I. Cardiac infarction. II. Recent chole cystectomy.
45. Simmons, William H.	05-05-69 PAB	43	4109 I. Acute myo infarct. ASHD with angina pectoris. II. Emphysema.
46. Stover, Enoch	09-28-73	74	4109 I. Cardiac standstill. Massive myo infarct.
47. Swann, Dillard C.	07-07-69	47	1621 I. Carcinomatosis. Squamous cell cancer right lung with metastasis to both lungs, liver, and lymph nodes.
48. Terry, Willard B.	06-20-80	59	4109 I. Acute myocardial infarction: Arteriosclerotic heart disease.
49. Tucker, Russell S.	02-19-73 PAB	50	1621 I. Bronchiogenic carcinoma with metastasis.
50. Vintroux, James A.	04-16-57	42	9240 I. Severe burns (chemical explosion).
51. Waugh, Lawrence	08-04-79	60	5340 I. Cardiac arrest. G.I. bleeding, massive recurrent peptic ulcer disease. II. Congestive heart failure, severe.
52. Withrow, Corbett	04-29-74	49	1850 I. Transitional cell carcinoma of prostate with metastasis.

Note: Death certificates attached.

Chloracne Cases 1950-1954 Deceased

	Name	Date of Death	Age	Cause of Death
53.	Wolfe, Leslie	On Group I list.		5193 I. Acute respiratory arrest. COPD.
54.	Workman, James L.	02-11-79	86	4121 I. Cardiopulmonary arrest. Hyper. ASHD.
55.	Workman, John L.	08-30-71	50	1519 I. Carcinoma stomach.
56.	Young, Otis Henry	03-07-69	69	4109 I. ASHD, recurrent acute myo infarct. Intractable CHF. II. Ileac, pneumonia.

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Note: Death certificates attached.

GAFFEY V. MONTAGUE

TIMELINE OF SIGNIFICANT EVENTS RELATING TO ZACK-GAFFEY FRAUD

Overview:

Orginal proposal: 1 study to follow up on Suskind's original 37 workers studied in early 50's.

Next step: broaden study to avoid bias that might be introduced by focusing on most severely afflicted !!!!

Next step: can't just study morbidity because many workers have died, therefore mortality must be studied as well

Next step: Mortality is broken in two, one for accident-exposed and one for process-exposed

Therefore: 1 Morbidity study = Suskind-Hertzberg
1 Mortality study to study accident-exposed "chloracne" workers only
1 Mortality study to study all process workers (which presumes effects might show without chloracne, which Suskind can not sign his name off on).

Jury should be allowed to infer fraud from change in study design for Zack-Gaffey from that announced only seven months previously in Zack-Suskind; therfore, it's critically important to get the data from missing chloracne study and all records relating to decisions to change study designs. See Kemner Exhibit 393!!!!! (the missing cohort).

1976 ICMESA plant in Seveso, Italy has reactor vessel explosion similar to 1949 accident at Nitro, W.Va. Profound and obvious acute effects on humans and wildlife.

1976 Suskind writes Monsanto proposing follow-up on 37 workers he studied in early '50s in light of Seveso accident; no indication of acceptance by Monsanto

1978 Agent Orange Vietnam Veterans file class action

1978 August 11. Kemner Ex. 76. Monsanto document discusses Agent Orange, Seveso, Suskind proposal, recommends that study be performed, but be enlarged to avoid "bias" resulting from Suskind's original group possibly having been the most severely afflicted workers. ROUSH received exhibit.

1979 EPA does emergency suspension of some 2,4,5-T registrations because of spontaneous abortions in Western

Oregon

1979 Suskind and Monsanto confer again, agree that study is needed in light of Agent Orange class action, Seveso, and EPA emergency suspension of 2,4,5-T; study of Nitro workers begins but not expedited.

1979 January 24. Kemner Ex. 63. Union announces it has retained Mt. Sinai (Selikoff-Moses) to do study of Nitro workers to be conducted on behalf of workers' union. Document mentions phone call from Suskind saying he had been requested by Selikoff to assist in Mt. Sinai study. Monsanto proposes hiring Suskind themselves to do "objective" study to counter "anti-business" Selikoff. Document states that Suskind isn't anti-business). ROUSH is mentioned in document, but not as recipient.

1979 April 16. Kemner Ex. 68. Judith ZACK letter to ROUSH and others, discussing meeting with Suskind to discuss Suskind's "upcoming medical examination study" of Nitro workers and Suskind's information needs. Preliminary definitions of study cohorts. Suskind wants to include all plant employees who worked between 1948 and 1969, excluding people from study "if an exposure classification could not be made." First appearance of plans to conduct mortality analysis, which Suskind wants to use same exposure classifications as morbidity study.

1979 April 18. Kemner Ex. 69. ZACK letter to SUSKIND, enclosing numerical summary of people in cohorts. Note that as planned at this stage, morbidity study would include maintenance workers and workers <u>who did not</u> contract chloracne.

1979 July 9. Nitro plant update (2,4,5-T\dioxins). No exhibit number. Memo by F.C. Meyer. Mentions EPA emergency suspension, Agent Orange, Seveso as impetus for studies. Preliminary results of Zack-Suskind show no effect, but stresses need for complete mortality experience and current health studies are completed. Important document, no witness scheduled other than MONSANTO 30(b)(6).

1981 December 28. Kemner Exhibit 393. The missing study cohort!! Janet YUNG memo to W.R. GAFFEY. Describes method of selection and attaches death records for the missing cohort of process-exposed chloracne cases, exposed 1950-1954. This is apparently the cohort of process-exposed chloracne cases described in the Zack-Suskind study.

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ADDENDUM TO PAT'S REPORT

(Unless otherwise noted, all references are to page and exhibit numbers in Pat's report.)

CHANGE IN STUDY DESIGN: THE MISSING COHORT

The Zack/Suskind mortality study of 1949 explosion workers with chloracne states (p. 11): "The results of this study will be incorporated with those of a larger study which will include plant workers exposed in the course of 2,4,5-trichlorophenoxyacetic acid production during the period 1948-1969," and (p. 12) "An analysis of the chloracne cases and exposures not associated with this accident but rather with the normal TCP/2,4,5-T production processes will be the subject of a future paper." (2,4,5-T was produced at Nitro from 1948 through 1969.)

The Zack/Suskind study announcing the above future study of TCP production workers with chloracne was published in January, 1980. Ten months later, in October 1980, Monsanto issued a press release proclaiming completion of the second study, identifying it as authored also by Zack and Suskind. (Monsanto medical director George Roush has identified this second study as the one subsequently published as the Zack/Gaffey study. pp. 437-438.) By the time of the October 1980 press release (pp. 347-350), however, the study described in the Zack/Suskind study was no longer a study of TCP workers with chloracne, and indeed did not address chloracne at all; instead, it had become a study of mortality in the entire Nitro plant, compared with mortality in a subset of TCP workers identified solely from employment records without any reference to medical condition (i.e., chloracne). The study differed from the study described in the Zack/Suskind study in several significant areas:

1. It did not address chloracne at all;

2. It eliminated from the study altogether all workers who died or ended employment at Nitro from 1948 [first year of 2,4,5-T production at Nitro] to the end of 1954, and from the end of 1977 to the end of 1978 [the cut-off for the Zack/Suskind study], and thus does <u>not</u> include "plant workers exposed in the course of 2,4,5-trichlorophenoxyacetic acid production during the period 1948 to 1969" as described in the Zack/Suskind study.

3. It included only hourly payroll employees within the above time limits and excluded from study all salaried employees (thus excluding 14 deaths of men with chloracne, according to Monsanto's own records, p. 339).

4. "Exposure" to TCDD was determined with no reference to

plant medical or safety records but solely by plant employment records (i.e., only workers in the actual TCP process), excluding all others (such as maintenance workers) who had potential exposure and leading to blatant misclassification of four workers classified as exposed in Zack/Suskind but as unexposed in Zack/Gaffey.

5. "Exposure" was determined only for a subset of dead workers, not for all TCP process workers (even by the limited employment criteria).

Thus, at some point between publication of the Zack/Suskind study and Monsanto's press release, the study design for the second mortality study had changed radically. No explanation has ever been given for this extreme change; the only reason given -- that no work history records existed prior to 1955 -- in no way explains why the study planned as a chloracne study was changed to a "work history" study in the first place. Dr. Roush testified that the two studies were begun and conducted at the same time (1978-1979, p. 443); at the time of publication of Zack/Suskind the second study was still a chloracne study, yet ten months later it had been transformed totally. What happened to the chloracne study?

A series of memos from Jan Yung, the person who collected the data for both studies, shows that the data for the second chloracne study (of production process exposure) was indeed collected. A December 5, 1978 memo from Yung (Tab 15 * to be numbered) summarizes sources for the 114 "dermatitis cases" in "Group I" (the accident exposures), and refers to the need for further work "to determine total dermatitis cases for Group II" (normal process exposures). (Tab 15 *)

[VERY INTERESTING NOTE: Yung refers to "the S.R.I. visit to computerize work histories at Nitro." I believe "S.R.I." is the Stanford Research Institute. Gaffey worked at SRI from 1976-1979: <u>is it possible he was involved in SRI's visit to Nitro and/or the computerization effort during 1978?</u> Good question for discovery/deposition. If he was, then he could actually have been working on the "work history" study <u>before</u> he came to Monsanto, which would explain how the study design could have gotten changed so radically in so short a time.]

A December 28, 1981 memo from Jan Yung to Gaffey establishes that a second cohort (Group II) was in fact identified from plant records ("safety, personnel, and medical records") and Workmen's Compensation records for the years 1950-1954. (Tab 16*) From this period, "a total of 135 white males were identified as having reported dermatitis or chloracne associated with TCDD exposure during normal TCP operations at the plant." Yung notes that "All of the individuals in the cohort were traced for vital status." As of the date of the memo (12/28/81), "we have a total of 56 known deceased with death certificates on file." Attached to her memo is the list of persons with chloracne associated with

normal TCP operations, and a table of "Chloracne Cases 1950-1954 Deceased" listing names, date of death, age, and cause of death. (Of these, roughly 42 died of heart/circulatory disease, and 11 of cancer; of the heart and cancer deaths, 20 were at or before age 50; of these 20, 6 were cancers. These are rough numbers, but do suggest that men with chloracne were dying of heart disease before the opportunity for cancers to develop.)

Significantly, this 1981 memo identifies a TCP-process chloracne cohort exposed only from 1950-1954 (although the vital status is given as of Dec. 1981). The Zack/Suskind study promised a cohort of process workers exposed from 1948 through 1969. Was a cohort for this entire period identified? Testimony in the Nitro case was that there were chloracne cases occurring during every year of this period (Nitro transcript, pp. ___*). Apparently Yung wrote the memo in response to a request from Gaffey for data only on 1950-1954. If the data for the entire period (through 1969) reflect the trends in the 1950-1954 period, that is probably why Monsanto chose to change the study design to a work history study!

DR. SUSKIND'S RETREAT

Why Dr. Suskind refused to be named as author of the second study is still unanswered. Given his career-long dedication to chloracne as the hallmark of TCDD exposure, he may simply have been angry at his study design being changed. He may also have recognized the misclassifications in the changed study and would not put his name to a study that classified as unexposed the very people he had classified as exposed in the first, already published study. In addition, however, between 1949 and 1953 Suskind himself had examined and diagnosed with chloracne some of the very workers listed as unexposed in the Zack/Gaffey study.

Dr. Suskind is identified by Monsanto in its October 9, 1980 press release as the co-author of both the Zack/Suskind "accident" study, and what was later published as the Zack/Gaffey study. Dr. George Roush, Monsanto Medical Director, confirmed that the second "Zack/Suskind" study referred to in the press release and in the first Zack/Suskind is the Zack/Gaffey study (p. 437-438).

The question never answered by Dr. Roush or Suskind in the Kemner record is why Dr. Suskind took his name off the study. A clue may be found in the misclassifications and omissions of the Zack/Gaffey study. Among the individuals either misclassified as "unexposed" or excluded altogether from the Zack/Gaffey study are a number of men whom Dr. Suskind examined in 1953 and unequivocally diagnosed as exposed, based on his clinical exams and on Monsanto's own records.

The list of deaths in workers examined in 1953 (plaintiff's exhibit 1748) is at page 14 of text.

Monsanto's own record of misclassifications and exclusions from the Zack/Gaffey study is in Marcie Strauss's memo (pp. 336-341).

Of the four men classified as exposed in the Zack/Suskind study but as unexposed in the Zack/Gaffey study, one (Ralph Westfall, p. 336) was diagnosed by Suskind in 1953 (p. 14 text); of 14 men included as exposed in the Zack/Suskind study but excluded altogether from the Zack/Gaffey study, three (John E. Blackwell, Rarry Hudnall, and Lawrence L. Shank, p. 339) were diagnosed as exposed in 1953 (p. 14 text); and of three men (p. 341) listed as "unexposed" in Zack/Gaffey who should have been exposed, one (William Simmons) was diagnosed as exposed in 1953 (p. 14 text).

Thus, Suskind was unquestionably aware that the change in study design for the second study, from a chloracne study to an "hourly worker" study, not only resulted in misclassifying workers clearly classified as exposed in Zack/Suskind, but also excluded workers he had diagnosed -- and Monsanto had identified -- as exposed in 1953. This perhaps was too much even for Dr. Suskind to put his name to.

Suskind testified that Monsanto did not want to include citations to his 1949, 1950, and 1953 unpublished studies in the Zack/Suskind study (NITRO transcript 28590). Monsanto's motives in wanting to keep those citations out may have been to prevent readers from discovering what Suskind knew. (Judith Zack's argument that the Journal of Occupational Medicine had a policy of not citing unpublished data [NITRO P. 28593] is pretty weak.) The actual records cited on these NITRO transcripts would be important to obtain. We don't have them.

AUTHORSHIP OF ZACK/GAFFEY STUDY

1. Gaffey states in his answers to interrogatories that:

   A. He came to work at Monsanto in 1979 [p. 3]

   B. He initiated the Zack/Gaffey study and it was approved by his superior Dr. Roush [p. 8]

   C. Dr. Roush was his immediate supervisor [p.3]

   D. The present custodian of the data and documents used in preparing the Zack/Gaffey study is James J. Collins, Epidemiology Director, Monsanto Company.

2. There are strong indications, however, that Gaffey in fact was not involved in the actual conduct of the Zack/Gaffey study at all, and was not named as an author until after the study was completed.

A. Dr. Roush testified that the work for the Zack/Gaffey study was begun in 1978, "about the same time" as the work for the Zack/Suskind study [p. 69].

B. Jan Yung was in Nitro, W. Va., in November, 1978, collecting data for both Group I (1949 accident cases) and Group II (non-accident exposure). [pp. 99-89] Marcie Strauss confirms that Yung developed two lists: "the 'Nitro TCP Accident' list with 122 people on it and the list of 'Chloracne Cases Not Related to 1949 TCP Accident' containing 228 people." [p. 18]

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C. Gaffey did not come to work for Monsanto until 1979 [p.3], and therefore could not have either "initiated" the study nor participated in the work done on it prior to his arrival at Monsanto. Gaffey is not listed as an attendee of the meeting of the Nitro Health Study Task Force on July 20, 1979 [pp. 80-82], but was a recipient of the minutes of that meeting [p. 80], at which the plan to issue a Monsanto press release on the Zack/Suskind study was discussed.

D. Monsanto's October 9, 1980 press release on the two Nitro mortality studies identifies both as being authored by Zack and Suskind [pp. 84-87]. Dr. Roush, testifying in the Kemner case, confirmed that the second study referred to in the press release is the Zack/Gaffey study. [p. 36] Dr. Roush acknowleged that the study originally identified by the press release as a Zack/Suskind study ultimately was published as the Zack/Gaffey study [p. 36] and testified that he didn't know why Suskind had removed his name from the study [pp. 36-38]

3. A copy of what was subsequently published as the Zack/Gaffey study was entered in the record of the 2,4,5-T cancellation proceedings on February 9, 1981. The study, dated August 14, 1980, and labeled "Draft," has only one author named, Judith Zack. Gaffey's name appears nowhere on it. Otherwise, it is the same study as was eventually published as Zack/Gaffey. Note that the date on this draft was only two months before the Monsanto October 9, 1980 press release.

4. If Gaffey in fact did not do the actual work on the Zack/Gaffey study, and is not in possession of the actual records used to conduct it, he has no basis for asserting its validity or the absence of fraud.

5. The only indication that Gaffey may indeed have been involved with the study is Jan Yung's reference to SRI's (Stanford Re-

search Institute) visit to Nitro to computerize work history records in 1978. (p. *___) Gaffey was head of SRI's epidemiology division at that time, and as a statistician would more than likely have been involved in the Nitro work history computerization effort. The dramatic and unexplained change from the planned chloracne study to a work history study, and its short-order completion, could have been Gaffey's work: having seen the horrific data on chloracne cases, Monsanto hired Gaffey away from SRI specifically to manipulate the computerized work history data (i.e., eliminating salaried workers, shortening time period, eliminating maintenance workers, etc.) to come up with an "acceptable" result. This could have been accomplished very quickly with the software available at that time. (This issue need not be explored yet, however, because the unanswered questions about Gaffey's authorship, as well as his admitted lack of any records, raise a sufficient question for summary judgment.)

OTHER ISSUES

1. Dr. Roush testified that the Zack Gaffey study is the study identified in the Zack/Suskind study as a forthcoming study of TCP process workers exposed to dioxin [pp. 63-64], and acknowleges that the two studies are "tied together." [p. 31] Monsanto's October 9, 1980 press release characterizes the Zack/Gaffey study as a "larger, follow-up effort" to the Zack/Suskind study. [p. 85]

2. The forthcoming study described in Zack/Suskind is described as a chloracne study [p. 63], however, while the Zack/Gaffey study did not involve chloracne at all [p. 69].

3. Dr. Roush (Gaffey's boss) admits that the Zack/Gaffey study deliberately classified workers exposed to dioxin as "unexposed", and that this was done "on purpose" because "of the design of that study" [p. 40]

4. How the design of the Zack/Gaffey study, described in the Zack/Suskind study as a chloracne study, changed into a study designed "on purpose" to classify workers with chloracne as unexposed to dioxin, is a critically important question in determining whether the Zack/Gaffey study was fraudulent.

Importance of Deposing Mary Gaffey

I. Summary

Mary Gaffey was supervisor of Monsanto's Corporate Environmental Information Center (medical library), the library which served the Medical Department, until 1989. She was likely initially employed in this position in 1979, when her husband William Gaffey was hired by Monsanto, and she retired in 1989, the same year William Gaffey also retired from Monsanto.

The medical library Mary Gaffey supervised served the Department of Medical and Environmental Health (DMEH), in which William Gaffey and Judith Zack, authors of the Zack/Gaffey study, were employed, as well as Jan Yung, who collected data for both the Zack/Suskind and Zack/Gaffey studies. The library contained scientific journals, government documents, toxicology reports, and miscellaneous correspondence; the toxicology reports included both published reports and unpublished "proprietary" Monsanto reports. It is extremely likely, therefore, that this library, under Mary Gaffey's supervision, served as a resource for William Gaffey, Judith Zack, and Jan Yung in preparation of the Zack/Gaffey study.

Because the library was also a repository for medical department (including DMEH) correspondence, it is likely that correspondence relating to the Zack/Gaffey study and its conduct was maintained in the library under Mary Gaffey's supervision. Because all the library's correspondence files prior to 1985, which would include contemporaneous correspondence relating to the Zack/Gaffey study, was microfilmed and shipped off to storage in 1985, it is also likely that Mary Gaffey has knowlege of the present whereabouts of such correspondence and its contents.

II. Documents

A. Jacobson [Tab 21]

1. Until 1989, Mary Gaffey was supervisor of Monsanto's Corporate Environmental Information Center (medical library). [p. 10-12]

2. Mary Gaffey is married to William Gaffey [12-14].

3. The medical library contained journals, government documents, toxicology reports, and miscellaneous correspondence [12-14]

4. The library contained both published and proprietary (i.e., not published, not reported to government agencies) literature [14-17]

5. The library served the Department of Medical and

Environmental Health, within the medical department [19-21, 21-23]

6. The library's correspondence files prior to 1985 "were microfilmed and shipped off to storage" [32-34]

B. Marcie Strauss [Tabs 17, 18]

Strauss in 1984 prepared lengthy, documented critiques of the Zack/Suskind and Zack/Gaffey studies, identifying workers cross-classified and/or excluded. Strauss is in DMEH [see p. 1 of Tab 18, heading], and likely used the medical library to locate some or all of the records and correspondence she reviewed and appended to her critiques.

C. Jan Yung [Tabs 15 & 16]

Yung collected raw data for both the Zack/Suskind and Zack/Gaffey studies [Tab 14, p. 36, Zack deposition]. Yung was in DMEH [Id., see also heading on p. 1 of Tab 15, and p. 1 of Tab 16], and likely used the medical library to locate some of her materials.

D. Judith Zack [Tab 14]

Zack was co-author of the Zack/Gaffey study, was employed in DMEH from July 1977 to October 1981 [Tab 14, p. 4], and likely used the medical library in preparing her studies.

E. William Gaffey [Tab 12]

Gaffey, co-author of Zack/Gaffey study, was Epidemiology Director in DMEH [Tab 12, p. 3; see also Tab 16, p. 1, Gaffey and Yung both have identical mail code "G2WE" within DMEH]. He would likely have used the medical library in preparing the Zack/Gaffey study, and his correspondence was likely maintained in the library correspondence files.

C:\PROJECTS\MONTAGUE\AUTHOR

AUTHORSHIP OF ZACK/GAFFEY STUDY

1. Gaffey states in his answers to interrogatories that:

   A. He came to work at Monsanto in 1979 [p. 3]

   B. He initiated the Zack/Gaffey study and it was approved by his superior Dr. Roush [p. 8]

   C. Dr. Roush was his immediate supervisor [p.3]

   D. The present custodian of the data and documents used in preparing the Zack/Gaffey study is James J. Collins, Epidemiology Director, Monsanto Company.

2. There are strong indications, however, that Gaffey in fact was not involved in the actual conduct of the Zack/Gaffey study at all, and was not named as an author until after the study was completed.

   A. Dr. Roush testified that the work for the Zack/Gaffey study was begun in 1978, "about the same time" as the work for the Zack/Suskind study [p. 69].

   B. Jan Yung was in Nitro, W. Va., in November, 1978, collecting data for both Group I (1949 accident cases) and Group II (non-accident exposure). [pp. 99-89] Marcie Strauss confirms that Yung developed two lists: "the 'Nitro TCP Accident' list with 122 people on it and the list of 'Chloracne Cases Not Related to 1949 TCP Accident' containing 228 people." [p. 18]

   C. Gaffey did not come to work for Monsanto until 1979 [p.3], and therefore could not have either "initiated" the study nor participated in the work done on it prior to his arrival at Monsanto. Gaffey is not listed as an attendee of the meeting of the Nitro Health Study Task Force on July 20, 1979 [pp. 80-82], but was a recipient of the minutes of that meeting [p. 80], at which the plan to issue a Monsanto press release on the Zack/Suskind study was discussed.

   D. Monsanto's October 9, 1980 press release on the two Nitro mortality studies identifies both as being authored by Zack and Suskind [pp. 84-87]. Dr. Roush, testifying in the Kemner case, confirmed that the second study referred to in the press release is the Zack/Gaffey study. [p. 36] Dr. Roush acknowleged that the study originally identified by the press release as a Zack/Suskind study ultimately was published as the Zack/Gaffey study [p. 36] and testified that he didn't know why Suskind had removed his name from

the study [pp. 36-38]

3. A copy of what was subsequently published as the Zack/Gaffey study was entered in the record of the 2,4,5-T cancellation proceedings on February 9, 1981. The study, dated August 14, 1980, and labeled "Draft," has only one author named, Judith Zack. Gaffey's name appears nowhere on it. Otherwise, it is the same study as was eventually published as Zack/Gaffey. Note that the date on this draft was only two months before the Monsanto October 9, 1980 press release.

4. If Gaffey in fact did not do the actual work on the Zack/Gaffey study, and is not in possession of the actual records used to conduct it, he has no basis for asserting its validity or the absence of fraud.

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

BEFORE THE ADMINISTRATOR

In re:	
The Dow Chemical Company, et al.	FIFRA Docket Nos. 415, et al.

DIRECT TESTIMONY OF DR. RICHARD R. MONSON */

Scheduled Date of Testimony: October 1, 1980

Dorothy E. Patton
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*/EPA Exhibit No. 773

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

BEFORE THE ADMINISTRATOR

In re: The Dow Chemical Company, et al	FIFRA Docket Nos. 415, et al.

DIRECT TESTIMONY OF DR. RICHARD R. MONSON*/

I. Introduction

My name is Richard R. Monson. I am an Associate Professor of Epidemiology at the Harvard School of Public Health, Harvard University, Boston, Massachusetts. A copy of my curriculum vitae is attached to this testimony.

My testimony deals with my evaluation of several epidemiologic studies brought to my attention by the U.S. Environmental Protection Agency. These studies concerned the possible relationship between exposure to phenoxy acids and carcinogenicity. In general, the studies consisted of two types -- cohort studies and case-control studies. Both types of studies were designed to determine what relationship, if any, existed between the occurrence of certain types of cancer and exposure to phenoxy acids, including 2,4,5-T and its contaminant TCDD.

*/ EPA Exhibit No. 773.

My review, based on the general epidemiologic principles discussed below, focussed primarily on the studies conducted by Dr. Olav Axelson, Dr. Rainer Frentzel-Beyme, and Dr. Lennart Hardell. I also reviewed four other cohort studies involving phenoxy acids or TCDD. My general impression, based on these studies, is that although some methodological concerns are present in the studies, the results to date are consistent with the hypothesis of a causal relationship between exposure to phenoxy acids and/or TCDD and carcinogenicity.

II. General Observations

Epidemiologic data in human populations are generally derived from the observation of natural rather than experimental processes. The analysis and interpretation of epidemiologic data are therefore more complex than the analysis and interpretation of experimental data. Added complications are the relatively large time and space distances between cause and effect as compared to such distances in experimental settings. As a result, exposure and diseases cannot readily be measured in the same place at the same time. Further, there are usually no record keeping systems in general populations for routine measurement of exposures and diseases. For this reason, occupational groups are often used to assess any effects of chemical exposure on health.

Any review of an epidemiologic study must include evaluations of both the design of the study and the interpretation of the data. These areas are discussed separately below:

A. Study Design Considerations

1. Types of Studies

The cohort study is the usual type of study being done today to assess the frequency with which diseases occur in a given occupational setting. On the basis of personnel records the population of persons who have worked in a plant or an industry is defined. This past "cohort" of workers is then followed to the present, so as to determine which members of the population have died. From among the deaths, persons who have died from the disease under study (e.g. cancer) are identified. A comparison is then made between the disease mortality rate of these workers and the disease mortality rate of a comparison group.

Cohort studies may be either retrospective or prospective. In the context of the assessment of environmental pollutants, the retrospective cohort study is preferred. A group is defined on the basis of identifiable exposure at some time in the past, and the disease rate from that time to the present is measured. In prospective cohort studies a group is defined in the present and studied in the future. Such studies are less often performed since time must pass for new diseases to develop. In the case of diseases such as cancer with long latency or induction periods, this may be 20 or more years.

In assessing exposures associated with a given disease, the case-control type of study is performed. The investigator first identifies persons with the disease of interest. Such identification

generally takes place in a hospital setting. Next, a comparison group is identified. Such a group may be persons without the disease either in the hospital or in the general population. Case-control studies are generally not done solely for the purpose of evaluating the effects of a particular environmental or occupational exposure. Frequently, however, a person's occupation is one of the factors considered when looking at the past history of a group of persons with a specific disease.*/

2. Bias

"Bias" in general refers to a non-causal difference between exposed and non-exposed persons or a difference between diseased and non-diseased persons. Bias can lead to false associations between exposure and disease or can obscure true associations. One type of bias is observation bias, which refers to a difference in how data are collected from exposed and non-exposed persons or from diseased and non-diseased persons.

Observation bias results in cohort studies when the occurrence of disease in the exposure group is ascertained in a different manner from the occurrence of disease in the non-exposed group.

*/ There are two reasons why case-control studies provide only limited information on occupational exposures: (a) Usually, only one or two disease groups are studied at a time. Therefore, the picture one obtains as to the possible effects of the occupation is narrow. (b) Most occupations are uncommon. Therefore, even though a large case-control study is done, the number of cases (or of controls) who have worked in any one occupation is small. It is difficult to base conclusions on such studies.

While such bias is not consciously introduced by reputable investigators, it may be difficult to avoid.

Blindness in measurement of disease in a cohort study, or in measurement of exposure in a case-control study, will ideally prevent observation bias. If lung cancer is ascertained without knowledge of a person's occupation, no observation bias can result. However, if a person's occupation is known, lung cancer may be overdiagnosed in a specific group. Similarily, in case-control studies, observation bias could result if a person's lung cancer status is known when the occupational history is being taken. In both instances, an investigator aware of one factor and the proposed association may probe more vigorously for the second factor. If a blind study is not possible, an attempt is made to minimize observation bias by asking objective rather than subjective questions. (This is desirable even when blindness is possible). By so doing, greater uniformity of response may be achieved.

Selection bias results when persons who have both the exposure and the disease of interest are selectively entered into the study population. In a cohort study, selection bias may occur when a person's disease status is used in defining his exposure status. In a case-control study, selection bias may occur when a person's exposure status is used in defining his disease status.

3. Confounding Factors

In the assessment of the relationship between an exposure

and a disease, a confounding factor is a variable that leads to a non-causal association between the exposure and the disease. For example, if a certain group of employed persons are heavy smokers, an excess occurrence of lung cancer may be found in that group. The possibility exists that some investigators would conclude that the occupation produces excess lung cancer.

In this example, cigarette smoking is a confounding factor. It is associated with the occupation because the workers are heavy smokers and it is an independent cause of lung cancer. If smoking histories were available on persons in this occupational group, it would be possible to "control" for the confounding effects of smoking. One would look at the rate of lung cancer in smoking workers and in non-smoking workers relative to smoking non-workers and non-smoking non-workers. In this way the independent effects of smoking and employment can be assessed.

In experimental studies, confounding is usually not a major problem. Factors other than the factor being studied tend to be randomly distributed between exposed and non-exposed groups. Thus, any confounding factor that is itself also a cause of the disease being studied will tend not to be associated with the exposure of interest, because of the random occurrence of the confounding factors.

In non-experimental or epidemiologic studies, however, the process of exposure is not controlled by the investigator. As a result, there may be a tendency for two or more causes of a disease

to be associated. If the investigator is aware of which causes are associated, and can measure them, the effects of confounding can be minimized. Frequently, however, there are unknown causes of disease which lead to confounding. The investigator is unable to deal with such factors.

Because almost all data relating exposure and disease in human populations are non-experimental, the probable existence of unknown confounding makes the interpretation of epidemiologic data a difficult process. It is rare for one study or body of data to produce unequivocal evidence that a given exposure is (or is not) a cause of one or more diseases. Most epidemiologists, therefore, are cautious in interpreting associations based on one study. Replication is usually a necessary element in assessing causal relationships based on epidemiologic data.

B. Interpretation of Epidemiologic Data.

The interpretation of epidemiologic data is a complex topic. The following are some of the considerations which are involved in that interpretation:

1. Consistency

Consistency refers to the reproducibility of results in two or more studies. As indicated above, observation and confounding biases are frequent problems in non-experimental studies. To the extent that different investigators in different studies in different populations obtain similar results, the greater is the likelihood that the results are correct. While this is clearly a

judgmental issue, it is usual that different epidemiologists have different prior opinions, conduct studies in different ways, and study different groups. If, in spite of all these differences, similar associations are found, the belief in the general result is strengthened.

2. Strength of Association

The relative risk (RR) is the rate of disease in an exposed group divided by the rate in the non-exposed group. An RR of 1 indicates no association between exposure and disease. The higher the RR becomes, the more believable is the proposition that the association is causal. This does not mean that causal associations must have high RR's. Rather, it is a general opinion that undetected bias is less likely when the RR is high. That is, if some confounding factor is present, it is more obvious when it leads to a high RR than to a small one.

RR's less than 1.5 are difficult to assess using epidemiologic methods. The presence of methodologic biases and random variation make it difficult to detect causal associations against this background "noise". RR's of 3-4 or greater are large and are detectable by relatively crude studies. The range of approximately 1.5 - 3 is where epidemiologists have the most to contribute in the way of study design and data analysis.*/

*/ In mortality studies, standardized mortality ratios (SMR's) are frequently used rather than RR's. For practical purposes, an SMR equals 100 times an RR.

3. Statistical Significance

Caution must be exercised in using tests of statistical significance in the evaluation of data resulting from non-experimental studies. An association that is "statistically significant" may occur because of confounding, selection, or observation bias. In such instances, the formal "significance" has no meaning. Rather, a false impression is given that the association has a high probability of being causal. The conclusion that an association is not causal simply because it is not significant is equally misleading. This may only mean that there are insufficient data upon which to base an opinion. The most useful role of tests of statistical significance is in assessing the stability of the association seen in data. An association that is significant is stable; an assessment of the meaning of this stable association must take other considerations into account. An association that is not significant is not stable; other data are needed to assess the meaning of the association.

III. Evaluation of Cohort and of Case-Control Studies

In interpreting data from cohort studies and from case-control studies, one must always be concerned about observation, selection, and confounding biases. Only rarely can the data from one study be used to judge that an association is causal. In contrast, no number of studies can ever prove that an association is not causal, that is, that an exposure does not lead to some disease.

A. Cohort Studies

In prospective cohort studies, selection bias is not possible because the outcome of interest has not occurred when exposure is defined. In retrospective cohort studies, selection bias must be considered if persons have been enrolled in the study because of their disease status rather than because of their exposure status. Observation bias in cohort studies is a concern primarily in the assessment of outcome. Care must be taken that disease is ascertained in a similar manner in exposed and non-exposed groups. Confounding bias is essentially of equal concern in cohort and case-control studies. Any association seen can be due to confounding. In retrospective cohort studies, information may not be available on potential confounding factors. It then becomes a matter of judgment to assess the likelihood that an association may be due to confounding.

In retrospective cohort studies, random misclassification as to exposure is likely.*/ Exposure can rarely be measured with precision. Therefore, it is likely that in most retrospective cohort studies, on average, the estimates of relative risk that reflect causal associations tend to be underestimates.

*/ Random misclassification is another concern in epidemiology. Frequently, exposure has occurred many years in the past and cannot be measured with precision. Such imprecision can only blur any association between exposure and disease. That is, if a chemical causes a disease with a relative risk of 3, random misclassification of exposure will tend to minimize the measure of the association, that is, make it closer to 1.0. Random misclassification will never lead to the appearance of an association when none exists.

Retrospective cohort studies tend to have prospective aspects in that a cohort is defined on the basis of past exposure, is assessed today as to disease status, and continues to be followed into the future. The data available today will usually be inferior to the data that will be available in 5-10 years. At a minimum, more persons will have developed disease or will have died in the future, so that the data will be more stable. Of greater importance is the fact that assessment of a greater period of latency will be possible. If some exposure has been present only in the recent past, it may be many years in the future before any adverse effects become manifest. Thus, a study that is "negative" today may only reflect insufficient follow-up with respect to latency.

B. Case-Control Studies

Selection bias is a potential problem in most case-control studies. It is a special problem when cases are composed only of hospitalized persons. If there is a general belief that some exposure leads to some disease, diseased persons with the exposure may tend to be hospitalized in preference to diseased persons without the exposure. To the extent that all diseased persons in a defined population are enrolled into a study, selection bias becomes less of a problem. Observation bias is a special problem in case-control studies. Because information on exposure is frequently obtained directly from the cases and the controls,

differential reporting is possible. Blindness and objectivity must be emphasized to minimize this bias. Confounding bias, as in cohort studies, can always be invoked as a possible explanation for any association. Realistically, however, there are usually both positive and negative confounding factors that tend to cancel each other.

On occasion, "case-control studies within a cohort" are conducted. This usually refers to the case-control analysis of data that are collected in a cohort manner. Such "nested" studies are done to minimize the analytic costs. Also, one may have a cohort on whom it is expensive to obtain detailed exposure histories. In such a situation, exposure histories may be obtained only for cases of interest and for selected controls. The existence of a definable cohort is a requisite for such studies.

IV. Cohort Studies of Herbicide - Exposed Railway Workers.

I reviewed three papers prepared by Dr. Olav Axelson and his colleagues. (Axelson and Sundell, 1974; Axelson and Sundell, 1977; Axelson, et al., 1980). Exhibits 588, 590, 591. These papers detail Dr. Axelson's study of herbicide-exposed Swedish railway workers. This study was a cohort study, i.e., a group of individuals with a history of exposure to certain herbicides was followed over time to determine if any unusual disease patterns developed. Dr. Axelson was primarily interested in the development of cancer in this cohort.

In this study, three hundred forty-eight railroad workers who had worked for more than 45 days with herbicides between 1957-1972 were followed through October, 1978. The observed number of deaths for specific causes was compared to the number expected, based on Swedish national death rates specific for age, sex and calendar time. The data were stratified on whether the workers had been primarily exposed to phenoxy acids (2,4-D and 2,4,5-T), to amitrol (3-amino-1,2,4-triazol), or to a combination of the two types of herbicides.

The most relevant data in this study are those showing observed and expected numbers of cancers after a ten year latency period. (Table 1). That is, follow-up in this group did not begin until ten years after initial exposure to the herbicides. This was done because of the likelihood that any herbicide induced cancer would not lead to death until at least ten years after first exposure. The data are:

TABLE 1

	Number of deaths (observed/expected)			
Group	All causes	All cancers	Stomach Ca	Lung Ca
All	36/27.6	16/6.9	3/0.7	2/1.4
Amitrol only	4/7.8	3/2.0	0/0.2	1/0.4
Phenoxy only	17/12.5	6/3.1	2/0.3	0/0.6
Both Amitrol and Phenoxy	15/7.3	7/1.8	1/0.2	1/0.4

It can be seen that among those exposed to amitrol only, there is little to suggest an adverse effect from the exposure. However, among those with any exposure to the phenoxy acids, there is an excess of all cancer deaths combined and of stomach cancer. As indicated in the 1974 paper, other sites of cancer are represented by only one or two cases.

In my opinion, the data in this study are consistent with a hypothesis of an excess of fatal cancer among workers exposed to phenoxy acids, that is, among those exposed to phenoxy acids only or to both amitrol and phenoxy acids. Thirteen cancers were observed in this group after 10 years of latency, in contrast to the expected number of 4.9 derived from Swedish national death rates. The only specific site of cancer with an excess appears to be stomach cancer (3 observed, 0.5 expected). Because of the relatively small number of persons with cancer, and because of the apparent great diversity of types of cancer, judgement as to the meaning of the excess cancer is difficult. One can say with certainty, however, that this study offers no evidence to suggest that the use of phenoxy acids is safe.

V. Cohort Study of German Chemical Workers Exposed to Dioxin in 1953

I reviewed two manuscripts of a West German cohort study conducted by Dr. Rainer Frentzel-Beyme. (Thiess and Frentzel-Beyme, 1977; Frentzel-Beyme, 1979). Exhibits 536, 537. This investigation dealt with a cohort of chemical workers exposed

to TCDD after an industrial accident. The cohort was followed for 24 years to determine if any unusual disease patterns developed.

The study was based on a 1953 industrial accident which occurred in a plant where 1,2,4,5-tetrachlorobenzolin was converted to 2,4,5-trichlorophenol. Much of the building was contaminated and an attempt was made to decontaminate it. The building continued to be used between 1953 and 1968. Because it became apparent that the building remained contaminated, the structure was demolished in 1969.

The investigators established a cohort of 75 men of those who were exposed during the accident and during the subsequent cleaning and repair work. These men were followed for 24 years.

The mortality experience of the 75 men was compared to that of a matched comparison group from the factory, to mortality statistics for Rheinhessen-Palatinate (the state in which the plant was located), and to mortality statistics for West Germany. During the 24 year period, 17 men of the exposed cohort died and 11 of the nonexposed, or comparison, cohort died. There were 6 cancers, including 3 stomach cancers, among those exposed. There were no stomach cancers among the four cancer deaths in the comparison group.

On the basis of mortality rates for Rheinhessen-Palatinate, 15.3 deaths were expected in the exposed cohort. On the basis of mortality rates for West Germany, 16.0 deaths were expected. The

expected number of deaths from all cancer was 3.3 and from stomach cancer was 0.6.

For a study of this type, the size of the cohort used here is very small. The mortality rates for all causes, all cancers, and stomach cancer are elevated relative to the three comparison groups. Only for stomach cancer is the relative excess great. Since there were only 3 deaths from stomach cancer, the association is quite unstable.

The data in this study are, however, consistent with a hypothesis of an excess of stomach cancer among persons exposed to TCDD.

VI. Case-control Studies of Persons With Mesenchymal Tumors and Malignant Lymphoms

In addition to the cohort studies of Axelson and Fentzel-Beyme, I reviewed reports of several case-control studies conducted in Sweden (Hardell and Sandstrom, 1979; Eriksson et al. 1979; Hardell et al. 1980). Exhibits 594, 595, 596. The purpose of these studies was to compare the cases with an appropriate control group to determine whether the cases exhibited exposure patterns different from those in the controls. A case-control study is especially valuable where the disease under study is relatively rare.

I evaluated three case-control studies. Dr. Lennart Hardell observed between 1970 and 1976 seven patients with malignant mesenchymal tumors (soft-tissue sarcomas). Exhibit 765. All of these patients had been exposed to phenoxy acids as part of their work.

Subsequently, Dr. Hardell conducted a case-control study on 52 male patients with soft-tissue sarcoma and a 4:1 control group (Hardell and Sandstrom, 1979). Exhibit 594. For the 21 living cases, controls were selected from the general population and matched for age, sex and place of residence. For the 31 deceased cases, controls were selected from other deceased persons, except for cancers and suicide, and were matched for age, sex, year of death and place of residence. Cases and controls, or next-of-kin for deceased cases and controls, were contacted to determine the person's occupational history. For persons who stated a history of forest work or work in sawmills or the pulp industry, a questionnaire was sent to the individual's employer to ask about the use of phenoxyacetic acids or chlorophenols.

Of the 52 cases, 19 (37%) were determined to have been exposed to phenoxyacetic acids and/or chlorophenols in contrast to 19 (9.2%) of the 206 controls. The relative risk, defined as the disease rate among the exposed persons divided by the rate among the unexposed, was 5.7. The relative risks for exposure to phenoxyacetic acids only was 5.3, while that for exposure to chlorophenols only was 6.6. The relative risk for exposure to exhaust fumes was 0.8. 51% of the cases were smokers and 48% of the controls were smokers.

Eriksson et al., 1979, Exhibit 595, conducted a second case-control study of 110 men with sarcoma and of 219 controls. The study design was similar to that used by Hardell. The Hardell study was conducted in the north of Sweden and the Eriksson

study in the south of Sweden. Among the 110 cases, 25 (23%) were exposed to phenoxy acids and/or chlorophenols as opposed to 13 (6%) among the controls (relative risk = 5.1). For exposure to phenoxy acids only, the relative risk was 6.8. For exposure to chlorophenols only, the relative risk was 3.3. Exposure to asbestos, glass fiber, power saws, and smoking was relatively similar between cases and controls.

In a third study, Hardell et al. 1980, Exhibit 596, conducted a case-control study of 169 males with malignant lymphoma: 60 with Hodgkin's disease, 105 with non-Hodgkins' lymphoma, and 4 with unclassifiable lymphoma. For each case, two controls were selected, matched for vital status, sex, age and place of residence. Cases and controls were interviewed by questionnaires to determine the person's occupational history. Where data were unclear or incomplete, an interviewer not aware of the case/control status contacted the subject of the interview to clarify the data.

Of the 169 cases, 61 (36%) had been exposed to phenoxy acids or to chlorophenols in contrast to 32 (9.5%) of the controls (relative risk = 6.0). Among those with no exposure to chlorophenols, the relative risk for exposure to phenoxy acids was 4.8. Among those with no exposure to phenoxy acids, the relative risk for exposure to chlorophenols was 3.5 for low-grade exposure and 9.3 for high grade exposure. There was no evidence for any substantial confounding by a variety of other industrial exposures, including pesticides, or by smoking.

The authors state that analysis was made separately for two groups: Hodgkin's and non-Hodgkin's. Although no data are given, they state that no noticeable difference in the excess risk could be found.

In all three of these studies, relatively high relative risks were found for exposure to both the phenoxyacetic acids (primarily 2,4,5-T and 2,4-D) and to chlorophenols. The relative risks are based on substantial numbers of exposed cases and thus are statistically stable. In the absence of obvious methodologic faults in these studies, these high stable relative risks argue strongly for a causal association between the specific cancers and herbicide exposure.

One methodologic concern in these studies, as in many case-control studies, is whether exposure histories were obtained differently from cases than from controls. The strong association between herbicide exposure and sarcomas or lymphomas theoretically could have arisen if cases were questioned more closely or more extensively than controls. Similarly, the association could possibly have arisen if cases spontaneously over-reported or controls spontaneously underreported herbicide exposure. The authors were aware of these potential biases and seemed to do everything in their power to minimize them. However, one cannot say with certainty that at least some of the association might not be due to more careful questioning of the cases or spontaneous

under- or over-reporting of exposure by subjects.*/

Another possible, but unlikely, bias could have occurred in the selection of controls for the living patients. The cases were hospitalized; the controls were not. It is possible that the association is between herbicide exposure and hospitalization, not between herbicides and sarcoma or lymphomas. However, this bias could not be present in the case-control comparison of deceased persons. In the first study 60% of the patients were deceased and in the second study 35% were deceased.

Although I do have these concerns (i.e., there may have been methodologic differences in obtaining the exposure histories from cases and controls, and the association may be with sickness in general and not specifically with these tumors), I view it as unlikely that either of these concerns would have provided a major error in the data or in their interpretation.

VI. Other studies

In addition to the studies mentioned above, I also reviewed four other cohort studies of workers exposed to phenoxy acids.

1. Zack and Suskind, 1980, Exhibit 771, studied a 1949 industrial accident which occurred in the process of manufacture of 2,4,5-T from trichlorophenol (TCP). Among the workers exposed to TCDD during accident and the subsequent cleanup, 121 developed

*/ A repeat of the study using persons with solid tumors (e.g. bladder, intestine, lung) and with non-cancers (e.g. cirrhosis, nephritis, chronic respiratory disease, diabetes) would be valuable. If no association with herbicide exposure could be demonstrated for these diseases, the specificity of the association with the mesenchymal tumors and the lymphomas would argue strongly for a causal interpretation of the association.

chloracne. The mortality experience of this group from 1949 to 1978 was determined.

Thirty-two deaths occurred among this study group compared with the 46.4 deaths expected from age-time specific mortality rates for U.S. white males. Other observed/expected numbers included: all cancer (9/9.0), lung cancer (5/2.9), and lymphatic and hematopoietic cancer (3/0.9). The three lymphatic and hematopoietic cancers were Hodgkin's disease, lymphatic leukemia, and acute myelogenous leukemia. Also, there was one death from a soft-tissue sarcoma (a malignant fibrous histiocytoma).

This is a relatively small cohort study, and the resultant data are consequently unstable. There is no excess of death from all causes or from all cancers, relative to the U.S. mortality rates. However, on a proportional basis, there is an excess of cancer (28% observed, 19% expected). There are absolute excesses of lung cancer and of lymphatic and hematopoietic cancer. There is one death from soft-tissue sarcoma, a rare malignancy.

The data in this study are consistent with a moderate excess of lung cancer and of lymphatic and hematopoietic cancer after exposure to TCDD. The excesses are based on small numbers and are therefore unstable. The one case of soft-tissue sarcoma is unusual.

2. Between 1951 and 1971, another cohort of 204 men who worked at least one month in a 2,4,5-T manufacturing process were identified and studied by Ott et al., 1980. Exhibit 774. Of these men,

157 worked less than one year in operations where exposure to 2,4,5-T was measured. They likely were also exposed to 2,4,5-T and other chemicals in other areas for longer but unspecified periods of time. None of these men developed chloracne. The mortality experience of these men between 1951 and 1976 was examined.

Eleven deaths occurred in the study group and 20.3 were expected from the age-time-specific mortality rates for U.S. white males. One death from cancer (lung cancer) was observed and 3.6 were expected.

This is a very small cohort study, and the resultant data are very unstable. These data provide no support for the hypothesis that exposure to 2,4,5-T causes cancer in humans.

3. Riihimaki et. al., Exhibit 598, conducted a cohort study of 1960 men in Finland who sprayed brushwood-killing herbicides between 1955 and 1971. The cohort was followed through 1976. The observed numbers of deaths for specific causes were compared to the numbers expected, based on Finnish national death rates specific for age, sex and time.

Overall, the mortality rate from "natural causes" among this group was 50% of that expected (70 observed, 148.1 expected). For malignant neoplasms, 21 deaths were observed and 34.5 were expected. There was no apparent excess of death from any specific type or site of cancer.

There is no evidence in these data that herbicide-spraying is associated with death from any cause. The data differ sharply

from those in Sweden, which refer to roughly the same time period. While one could argue that, in this study, sufficient time had not passed to permit cancer to develop following exposure, the same argument would have to apply to the Swedish data. Differences in the selection criteria for the Finnish cohort (10 days versus 46 days of exposure in the Swedish study) could be another reason for the differences in the two sets of data.

4. Cook et al., Exhibit 772, conducted a cohort study of 61 males who in 1964 were exposed to TCDD in an industrial setting. Chloracne developed in 49 of these workers.

Between 1964 and 1978, 4 of these men died; three of the deaths were due to cancer: adenocarcinoma-primary site unknown; fibrosarcoma, and glioma with metastases. Based on U.S. mortality rates for white males the expected number for all causes was 7.8 and for all cancers was 1.6.

The overall mortality experience among this small group is considerably less than that of the general population (SMR = 51). However, there is both a relative and absolute excess of death from cancer (75% of deaths were from cancer as compared to an expected percentage of 21%). There is one death from a soft-tissue sarcoma, which is an unusual cause of death.

This study is consistent with the hypothesis that exposure to TCDD causes a fatal excess of cancer. The excess, however, is based on small numbers and is unstable.

VIII. Summary

There is no absolute process through which we can learn whether a chemical causes a disease. Different persons will collect, analyze, and interpret data differently. Errors will be made which are not detected. Conflicting results will occur. In the face of such uncertainty, it seems reasonable to err on the side of safety. If there is a suggestion that a substance is carcinogenic, it should be treated as such until further data are available.

The three case-control studies in Sweden strongly suggest that working with phenoxy acids and/or chlorophenols leads to soft-tissue sarcomas or lymphomas. The relative risks are relatively high and are based on stable numbers of cases. However, the ascertainment of exposure to herbicides in general and to 2,4,5-T and 2,4-D specifically was based on a subjective process that was potentially open to bias, although careful steps were taken to prevent that bias.

The cohort study of Swedish railroad workers is small, but shows a relatively high excess of death due to cancer. This excess is comprised of a variety of unrelated sites of cancer, although the excess of stomach cancer is consistent with the excess of stomach cancer in the German cohort study.

The German cohort study and the three U.S. cohort studies are based on small numbers of deaths and give unstable and ambiguous

results. There is no consistency of type of cancer among these studies, but there is an excess of stomach cancer in both the German and Swedish studies. Sarcomatous tumors may occur in the stomach. Further, in two of the U.S. studies, there is one death from a soft-tissue sarcoma, an unusual cause of death.

The levels of exposure in all of the studies are difficult, if not impossible, to assess. In three of the cohort studies, industrial exposure to TCDD was relatively high as indicated by the occurrence of chloracne. In the other studies, the level of exposure was presumably less but the accumulated length of exposure was presumably longer.

The inability to measure exposure accurately is the usual situation in epidemiologic studies with retrospective aspects (case-control studies or retrospective cohort studies). This inability usually is of more concern in studies where no overall association between exposure and disease is seen than in studies where a positive association is seen. In studies of "no association", it may be that there exists a relatively highly exposed subgroup with an excess of disease that has not been identified. However, if an association between exposure and disease is seen in spite of an imprecise measure of exposure, it only means that evaluation of any dose-response relationship may not be possible. The overall association is not affected by this imprecision.

In evaluating the picture presented by these studies, it must be emphasized that one can never prove that a substance is safe, e.g., that it does not cause cancer. At best, one can only present evidence that no excess of cancer has been demonstrated to result after exposure to the substance. Also, it is not necessary that all studies of a substance that does cause cancer be positive. There may be a number of explanations for a "negative" study of a substance that is carcinogenic: chance, bias, minimal exposure, insufficient time for the cancer to develop. One well conducted study based on stable numbers that shows a strong positive association between some exposure and some disease must be weighted much more heavily than a number of well conducted studies based on stable numbers that show no association between exposure and disease.

The three case-control studies show strong positive associations based on stable data. I am unaware of situations in epidemiology where stable relative risks of approximately 6 have been shown to be due to observation bias. In my opinion, observation bias can usually account for relative risks of perhaps 1.5 to 2.0. While it is not impossible that the stable relative risks of approximately 6 could be due to observation bias, I believe the authors were aware of the potential problem and took steps to minimize it.

Of the six cohort studies I reviewed, an unstable excess of cancer was seen in four. Soft-tissue sarcoma, an unusual cause of death, was seen in two. In two studies, an excess of cancer of the stomach was seen, a site in which sarcoma may occur. While no one of these cohort studies presents convincing data as to the human carcinogenicity of phenoxy acids, taken together they are consistent with the case-control studies.

On the basis of the Swedish case-control studies, I would behave as if 2,4-5-T and 2,4-D were carcinogenic in man. I would do additional case-control studies of other cancers to assess the likelihood that the association between sarcomas and phenoxy acids exposure was due to some methodologic bias. I would continue the cohort studies for at least 10-20 years to provide more stable mortality data.

Richard R Monson (4/00)

Richard R. Monson

EXHIBIT LIST

Exhibit No.

536a Thiess, A.M. and R. Frentzel-Beyme, 1977. Mortality of persons exposed to dioxin after an accident which occurred in the BASF on 13th November 1953. Paper presented at MEDICHEM Congress V in San Francisco, September 5-9, 1977.

536b Table: Cancer deaths of the BASF Dioxin study, by site and morphology of malignancy (1977).

537 Frentzel-Beyme, R., 1979. Revised draft of BASF dioxin study.

588 Axelson, O. and Sundell, L., 1974. Herbicide exposure, mortality and tumor incidence: An epidemiological investigation on Swsedish railroad workers. Work Environ. Health 11: 21-28.

590a Axelson, O., and Sundell, L., 1977. (In Swedish) Fenoxisyror och cancer. Lakartidningen 74: 2887-8.

590b EPA translation of Axelson and Sundell, 1977.

591a Axelson, O., Sundell, L., Andersson, K., Edling, C., Hogstedt, C., and Kling, H., 1980. Herbicide exposure and tumor mortality; an updated epidemiological invesigation on Swedish railroad workers. Scand. J. Work Environ. Health 6: 73-79.

591b Axelson, et al., 1980, pre-publication manuscript.

594 Hardell, L. and Sandstrom, A., 1979. Case-control study: Soft-tissue sarcomas and exposure to phenoxyacetic acids or chlorophenols. Brit. J. Cancer 39: 711-717.

595a Eriksson, M., Hardell, L., Berg, N., Moller, F. and Axelson, O., 1979. (In Swedish) Case-control studie over maligna mesenkymala mjukdelstumorer och exposition for kemiska substanser. Lakartidningen 76: 3872-75.

595b EPA translation of Eriksson et al., 1979.

596a Hardell L., Eriksson, M. and Lenner, P., 1980. (In Swedish) Maligna lymfom och exposition for kemiska substanser, sarskilt organiska losningsmedel, klorfenoler och fenoxisyror. Lakartidningen 77: 208-210.

596b EPA translation of Hardell et al., 1980.

598 Riihimaki, V., Asp., S., Seppalainen, A.M., and Hernberg, S., 1978. Symptomatology, morbidity and mortality experience of chlorinated phenoxyacid herbicide (2,4-D; 2,4,5-T) sprayers in Finland. A clinical and epidemiological study. Working paper presented at the National Institute of Environmental Health Sciences and International Agency for Research of Cancer Conference on the Long-term Hazards of Polychlorinated Dibenzodioxins and Polychlorinated Dibenzofurans, Lyon, France, January 10-11, 1978.

765a Hardell, L. 1977. (In Swedish) Maligna mesenkymala tumorer och exposition for fenoxisyror - en klinisk observation, Lakaridningen 74: 2753-4.

765b EPA translation of Hardell 1977.

771 Zack, J.A. and R.R. Suskind, 1980. The mortality experience of workers exposed to tetrachlorodibenzo-dioxin in a trichlorophenol process accident. J. Occup. Med. 22: 11.

772 Cook, R.R., J.C. Townsend, M.G. Ott, and L.G. Silverstein, 1980. Mortality experience of employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). J. Occup. Med. (in press).

774 Ott, M.G., Holder, B.B., Olson, R.D., 1980. A mortality analysis of employees engaged in the manufacture of 2,4,5-trichlorophenoxyacetic acid. J. Occup. Med. 22: 47-50.

CERTIFICATE OF SERVICE

I hereby certify that copies of the foregoing DIRECT TESTIMONY (AND EXHIBIT*) were hand delivered or mailed first class postage prepaid on September 17, 1980 to the following persons.

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1 page (cover sheet only)
Sent on 01-Nov-94 at 18:40

Comment:

NTIS will be Fedexing you PB91-107961, "Dioxin Registry Report of Monsanto Company, Nitro, West Virginia." It will arrive by Fedex tomorrow (if it's in stock) or Thursday (if they have to print you a fresh copy). Their computer shows one copy in stock but they don't trust their computer, so they're not sure. --Peter

Chemical company accused of hiding presence of dioxins

BY JOCK FERGUSON
The Globe and Mail

Monsanto Co., the giant U.S. chemical maker, knowingly hid the presence of dioxins in some of its products from the Canadian and U.S. governments, testimony in a suit against the company says.

The company found small amounts of the most toxic form of dioxin 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in its chlorinated phenol products — including Santophen, a germicide, and 2,4-dichlorophenol, which was made into a weed killer — when it started testing its products after an accidental spill in 1979.

Santophen was widely used as a disinfectant by hospitals and was the active ingredient in the widely sold household disinfectant, Lysol.

The maker of Lysol was not told by Monsanto of the presence of as much as three parts per billion of toxic dioxin in Santophen, according to evidence from company executives in a suit against Monsanto filed in East St. Louis, Ill.

The maker of Lysol stopped using Santophen in 1983.

Documents from the trial given to The Globe and Mail by Ross Harvey, NDP MP from Edmonton East, show Monsanto misled the Canadian government about the presence of TCDD contamination in Santophen, which it exported to Canada.

In a Sept.3, 1981, letter to a Health and Welfare Canada task force studying chemical safety, Monsanto said Santophen made between 1979 and 1980 showed no "evidence of the presence of 2,3,7,8-tetrachlorodibenzodioxin."

However, the company had evidence of 2,3,7,8-dioxin contamination in Santophen when the letter to Canada was written.

James Wilson, the company's manager of research and development, admitted in 1985 his letter to the Canadian government "was not correct ... I was mistaken obviously in writing that sentence."

Mr. Harvey is investigating how much of the contaminated germicide was imported into Canada from Monsanto's Krummrich plant in East St. Louis.

The 3½-year trial, the longest jury trial in U.S. history, ended in October, 1987, with the jury awarding punitive damages against Monsanto of $16.25-million. The company is now appealing the verdict and questions about its behavior have surfaced again.

Rex Carr is a lawyer for the plaintiffs, who claimed they were injured by the accidental spill of a dioxin-contaminated Monsanto chemical. In a brief to the Illinois Appeal Court, he says the trial transcript "contains literally hundreds of admissions that Monsanto was selling dioxin-contaminated chlorophenol products ... for 30 years, and that it did so with the knowledge that these products contained a contaminant that was highly toxic to both the environment and to human beings."

David Snively, a lawyer for Monsanto in St. Louis, said what Mr. Carr alleges is a very selective interpretation of company records and the testimony of its executives during the trial.

Dan Bishop, a spokesman for Monsanto in St. Louis, said the company is moving to strike parts of Mr. Carr's brief as "grossly misleading and inaccurate."

The evidence of Monsanto executives at the trial portrayed a corporate culture where sales and profits were given a higher priority than the safety of products and its workers.

In the late 1970s and early 1980s company executives decided not to tell customers about the dioxin-contamination problems for fear of losing sales.

Company records show the presence of dioxins as high as 20,000 parts per billion in its chlorinated phenol products, yet it never notified the U.S. Environmental Protection Agency of possible hazards to users, Mr. Carr said.

Monsanto says that the jury in the trial found that none of the plaintiffs had been injured in the spill. Damages were awarded against the company only because of Mr. Carr's allegations, Mr. Snively said in an interview.