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EXHIBIT #1

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TOXIC AND TERATOGENIC EFFECTS OF 2,4,5-TRICHLOROPHENOXYACETIC ACID AND 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN

Summary of Evidence Presented to The Royal Commission Hearings On Herbicides and Pesticides, July 16, 1974 and to Hearings of the Assembly Committee on Natural Resources of the State of Wisconsin March 19, 1975

by

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Materials presented here came from the study of published and unpublished reports of experiments on the effects of 2,4,5-T and related products, among them reports submitted by Dow Chemical and Hercules Inc. to the U.S. Food and Drug Administration, and to the National Academy of Science's Advisory Committee on 2,4,5-T, to the Administrator of the Environmental Protection Agency, and from a study of briefs submitted to the courts by attorneys for Dow Chemical, the National Environmental Defense Fund, and the Environmental Protection Agency.

ABSTRACT

The evidence is reviewed concerning the teratogenic and other toxic effects of 2,4,5-T and its tetra-dioxin contaminant, TCDD, including unpublished data abstracted from submissions by registrants to the U.S. Environmental Protection Agency. 2,4,5-T containing even minimal amounts of TCDD and in a "technically" pure state is teratogenic, embryotoxic and fetogenic and may well be mutogenic and carcinogenic. Contrary claims that 2,4,5-T containing .5 ppm or .1 ppm (or less) TCDD given in small quantities (less than 40 mg/kg per day) does not have teratogenic effects do not appear to be supported by evidence supplied by U.S. industrially sponsored research, and is contrary to conclusions submitted with these same data. Cumulative evidence makes it reasonable to conclude that the effects of 2,4,5-T are no different and certainly no less severe on man than on aminals. A false feeling of safety had been created by erroneous methods of quantitatively generalizing from animal experiments to what might possibly happen to man.

OUTLINE OF THE PROBLEM

Since 1970 there has been much discussion and much confusion about the evidence on toxic reactions to 2,4,5-T and to its tetra-dioxin contaminants. Some of the most relevant data have never been published in U.S. scientific journals. While much of these data may have been presented to different U.S. regulatory agencies, claims made about the meaning of these same data may really not have been justified. 2,4,5-T is widely used throughout the world and the issue raised by the possible teratogenic effects of this herbicide is exceedingly important. The full available evidence needs to be summarized for full public review.

We will review the evidence that 2,4,5-Trichlorophenoxyacetic acid (2,4,5-T) and/or its constant contaminant, 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) are exceedingly toxic in animals. 2,4,5-T containing minimal amounts of TCDD and in a "technically" pure state is definitely teratogenic, embryotoxic, and fetogenic, and may well be mutagenic and carcinogenic. Further, exposure to TCDD induces lethal and sublethal chronic health effects. It will be noted that the so-called "negative" evidence that 2,4,5-T is not teratogenic in relatively small doses is based largely on industry-sponsored experiments that are inadequately designed and which present data that are inadequately, and sometimes erroneously, analyzed.

The effects of 2,4,5-T on man may be no different and certainly are no less severe than the effects on experimental animals. Similarly, the effect of 2,4,5-T on the non-human environment may be just as damaging. It will be noted that in relating animal to human data a certain confusion has been created through erroneously generalizing from doses used in animal experiments to possible exposure levels in man.

A. EXPERIMENTAL EFFECTS ON ANIMALS OF 2,4,5-T AND/OR ITS CONTAMINANT, TCDD

The effects of 2,4,5-T are difficult to evaluate alone because various amounts of TCDD are almost always present in test samples.

TCDD is a Potent Teratogen and is Embryotoxic in the µg/kg Range

Two teratogenic effects have been clearly related to TCDD: cleft palate and kidney abnormalities. Frequent embryotoxic effects are intestinal hemorrhage, infiltration of fat into the liver, subcutaneous edema, and delayed ossification.[1] Increased fetal mortality, early and late fetal resorption, and intestinal hemorrhage of the fetus had been found in rats at a dietary dose of .125 to .1 µg/kg.[2] Kidney abnormalities have been produced in rat fetuses at the dose of .5 µg/kg TCDD.[3] The same study also reported cleft palate and kidney abnormalities in three strains of mice after injection with 1 to 3 µg/kg during the sixth to fifteenth day of pregnancy.

2,4,5-T is Teratogenic and Embryotoxic in "Pure" Form or at a Commercial Level That Contains No More Than .1 ppm of TCDD

A number of investigations have demonstrated that 2,4,5-T in its pure form or the form in which it is considered pure enough for marketing

is definitely teratogenic and embryotoxic. Samples of 2,4,5-T containing no more than .5 ppm of TCDD were teratogenic and embryotoxic in chick embryo studies.[4] The same investigator also demonstrated that extensively purified 2,4,5-T samples with no TCDD detectable with present techniques caused a significant incidence of embryotoxicity in the hamster and chick embryos and gross terata in the chick embryo.

The well-known study by Courtney and Moore using 2,4,5-T in dose per day of 100 mg/kg containing less than .5 ppm TCDD produced cleft palate and kidney malformations in three strains of mice.[5] In an unpublished and generally unknown repetition of the Courtney-Moore experiment, sponsored by Hercules Inc., (a former manufacturer of 2,4,5-T) Bionetics Research Laboratories repeated the same experiments using samples submitted by Dow Chemical and other registrants. The results were identical with that of the Courtney-Moore study. Developmental abnormalities included cleft palate, and abnormal leg positions.[6] At that time both Dow and Hercules claimed production of 2,4,5-T with .1 ppm or less of TCDD.

It has been shown by Collins and Williams that commercial samples of 2,4,5-T supplied by Dow Chemical (consisting of a "technically" pure preparation containing .5 and .1 ppm dioxin and a "pure" sample containing no detectable dioxin) and supplied by Hercules Inc. and Eastman Kodak Company (also containing no detectable dioxin) greatly increase fetal mortality in hamsters particularly in early embryos.[7] Also, an increased incidence of hemorrhage in live-borns was observed and there was an increase in the number of malformations among the live-borns. Dosage of 40 mg/kg of 2,4,5-T with no detectable TCDD caused decreases both in the percentage of viable fetuses and in the average fetal weight. Increasing

the amount of TCDD in the 2,4,5-T generally increased incidence of adverse effects in the hamster.

One more experiment that has not been widely reported was done by the Chairman of the Advisory Committee to E.P.A. in 1971.[8] Groups of pregnant Wistar rats were treated by gavage on the 9th day of gestation with doses of 100, 200, or 400 mg/kg and one dose was added of 20 mg/kg (but not on the 9th day, as in other groups, but spread from the 7th to the 13th day.) This experiment used questionable control groups. It is difficult to draw firm conclusions from its findings. However, percent dead or resorbed of litters and the mean weight of survivors in the group treated with 140 mg/kg and 100 mg/kg of body weight differed consistently from controls - as questionable as these controls might have been. (See Table I)

Other recent studies demonstrated that commercially pure 2,4,5-T can produce cleft palate in mice (at 35 mg/kg).[9] In another important study, cleft palate was induced in mice with 45 mg/kg of 2,4,5-T containing less than .02 ppm TCDD. As little at 50 mg/kg of purified 2,4,5-T and 12 mg/kg of 2,4,5-T Butyl Ester caused a decrease in fetal weight, which is a recognized sign of fetotoxicity.[1]

How About the "Negative" Evidence that 2,4,5-T Is Not Teratogenic at Low Doses of Exposure?

Experiments attempting to determine the dose-response relationship

Table 1

Embryotoxicity of 2,4,5-T Containing .5 ppm TCDD in Wistar derived rats (by gavage suspended in 0.2% carboxy-methyl-cellulose)

(From The Final Report of The 2,4,5-T Advisory Committee to EPA. This experiment was reported by the Chairman of The Committee, Dr. Wilson.[8])

Dose mg/kg	Days of treatment	Whole litters resorbed	Litters continuing to day 20			
			Total implants	% dead or resorbed	mean wt. of survivors	% survivors malformed[2]
Control[1]	9	0/45	558	7.2	3.8	0.8
20	7-13	0/11	170	8.8	3.7	0.7
100	9	0/11	170	9.0	3.7	1.9
200	9	0/11	156	11.5	3.8	5.1
400	9	2/10	122	25.4	3.2	11.0

1 *Cumulative vehicle treated control (per gavage) over past 4 years. The experiment had no control group as such, a circumstance that lead to some public discussion. The original data supplied by Wilson also used one other control for comparison, or so called "Cumulative untreated control over past 4 years." The meaning of that control or how it was actually obtained was never made clear. This control was omitted here. Also its inclusion would in no way modify our conclusions.*

2 *Types of malformations: anophthalmia, microphthalmia, curly or short tail, hydronephrosis, ectopic testes, agnathia.*

between 2,4,5-T and subsequent toxic or teratogenic effects have ignored many requirements of careful experimental design; specifically:

1. Inadequate attention has been paid to the requirements of sample size for treatment groups, especially groups that are treated with low levels of 2,4,5-T. The problem of adequate sample size (i.e. the sample size required to detect a particular effect) has been given thorough treatment in statistical literature.[11] Answers to questions concerning proper sample size depend on consideration of such things as the number of categories to be compared, the level of risk the experimenter is willing to assume, (i.e. the risk of a false positive and the risk of a false negative), and some reasonable guess at the size of the maximum standardized range of means to be observed. Toxicology studies that test for the dose effectiveness at low levels or are to be used to extrapolate to low levels of exposure are especially in need of sound experimental design. Yet we find that most experiments evaluating effects of 2,4,5-T simply allocate 5 to 25 animals per group (but usually 10 or so) regardless of whether the group was to be treated with 300 mg/kg of body weight per day or with 20 mg/kg of body weight per day. All of them use fewer animals than they should for results of treatment with low doses to be statistically valid.[12]

2. Data obtained from experiments are sometimes presented in raw form, in tables, and without proper statistical treatment.

The reviewer is often left to make sense of a lot of undigested numbers. Sometimes, too, observations that could be important either are not collected or not produced. However, when these data aggregates are examined, they indicate that 2,4,5-T is teratogenic at very low doses.

3. *Very often toxic or teratogenic manifestations that occur with low levels of treatment are ignored.*

In short, references to inadequate studies have created a myth -- that none of the mammalian species tested showed adverse effects at dosages of 40 mg/kg or less of maternal weight per day in response to 2,4,5-T.

Careful examination of the data does not warrant such optimistic conclusions, as a number of examples will show.

Emerson, Thompson, Gerbig and Robinson (from the Human Research and Development Laboratory of the Dow Chemical Co.) conducted an experiment in which rats were given doses of 1, 3, 6, 12 and 24 mg/kg of 2,4,5-T per day.[13] *Twenty-five animals per group were used. We immediately note that twenty-five animals is hardly a sufficiently large number to test the effect of a small dose at 1 to 24 mg/kg. Moreover the choice of rats rather than mice is specifically unfortunate, as mice are a much more susceptible model.*

The investigators then claim they failed to find evidence of teratogenic or embryotoxic effects. Yet, on inspecting their data we find a striking increase in the incidence of poor ossification of sternabrae, especially of the fifth sternabrae. (See Table 2) Incomplete and poor ossification, especially of the fifth sternabrae, has been reported by

Table 2

Number of Skeletal Abnormalities Observed In Pubs From Female Rats From Study by Emerson et al.,[13] (Abstracted From Table 6, page 13)

Skeletal Abnormality	*Control (Vehicle)*	*24 mg/kg of 2,4,5-T per day during organogenesis (days 6 through 15)*
Number of Animals	*103*	*103*
Accessory Ribs	*30*	*25*
Poorly Ossified 5th Sternebrae	*4*	*29*
Other	*0*	*5*

others in 2,4,5-T affected fetuses. Skeletal abnormalities are especially noteworthy because they were observed in another experiment using low doses of TCDD on the rat. This experiment by Sparschu, Dunn and Rowe (of the Biomedical Research Laboratory of the Dow Chemical Co.) on the effects of TCDD at low doses on the rat revealed skeletal abnormalities that were brushed off in the report as having occurred in all groups including the controls.[2] Yet the frequency of skeletal abnormalities is clearly dose-related in Sparschu's experiment. (See Table 3) Also noteworthy is that some fetal abnormalities occurred at doses as low as .03 mg/kg/day of TCDD and with considerable regularity at doses of .125 mg/kg/day. The data are at variance with the conclusions in the report submitted by Dow Chemical.

Although not related to the teratogenicity of 2,4,5-T, there is another curiosity in the Emerson report.[13] The investigators conclude that no adverse chemical effects were observed in treated dams during the period of treatment and gestation. Yet inspecting their data on the average body weight of females treated with 2,4,5-T it would appear that the control is consistently heavier than the treated groups. (Average weight in grams of females treated with 2,4,5-T from 1 mg/kg to 24 mg/kg was 343, 357, 349, 343, and 343 respectively and that of control groups was 359.) Quite contrary to the conclusions stated by Emerson, et al., their data do indicate both toxicity to the dam and teratogenicity to the offspring.

Another report by the same authors (again issued as a report by the Dow Chemical Company) is on a teratogenic study of 2,4,5-T acid using rabbits.[14] Four groups of twenty rabbits were tested at levels ranging

Table 3

Examples of Relation Between TCDD Dose to Mother And Some Skeletal Abnormalities From Sparschu et al.,[2] Given In Total Number Observed/Percent of Fetuses Examined (Abstracted From Table 8)

	Dose (g/kg/day) on Day 6 Through 15 of Gestation					
	Control	.03	.125	.5	2.0	8.0
Number of Fetuses Examined	93	42	45	37	3	-
Wavey Ribs	7/7.53	4/9.52	7/15.56	20/54.05	-	-
Incomplete Ossification						
Interparietal	8/8.6	3/7.1	10/22.2	9/24.3	3/100.0	
Parietal	5/5.4	2/4.8	5/11.1	12/32.4	2/66.7	
Frontals	0	0	2/4.4	1/2.7	2/66.7	
Occipital	2/2.1	0	0	0	2/66.7	
Nasal	0	0	0	5/13.5	2/66.7	

The category of "Poorly Ossified Sternebrae" is not shown since most fetuses examined, (including controls) appeared to have shown this sign.

from zero to 40 mg/kg of body weight. The investigators claim they did not find a dose-related trend evidenced in the parameters examined. Examination of their data does not bear out that claim. The weight of kittens is consistently less for the experimental than for the control groups. (40.2/mg for the control, and 33.2/mg, 33.4/mg, and 35.0/mg for the experimental groups). No statistical evaluation was made of these differences. However using an estimating procedure, the weight of control group is significantly larger than that of the experimental groups. (estimated $\overline{sx}$ = .805, $t = 7.86$ and $p < .01$ for 2 d.f.)

Although the investigators conclude that 2,4,5-T is not teratogenic or embryotoxic at doses up to and including 40 mg/kg/day, no detailed examination was conducted of kittens of groups that received 10 and 20 mg/kg/day. The only kittens examined were offspring of twenty females that were treated with 40 mg/kg/day and they in fact do show suggestive increases of skeletal abnormalities especially for accessory ribs.

The conclusions in this report by Emerson and associates again are at variance with the data and procedure in important ways. They failed to perform proper analysis and did not report noteworthy features of their data. We find it specifically disturbing that these investigators concluded that "Under the conditions of this study, 2,4,5-T was not embryotoxic or teratogenic at doses up to and including 40 mg/kg/day"[14, 15] when they had not even bothered to examine kittens of dams that had been exposed to 10 and 20 mg/kg/day. But even more convincing is the little known earlier study done by Bionetics for Hercules Inc. Following the first Courtney-Moore study, Hercules Inc. commissioned Bionetics to re-

peat that experiment with the same dose level of 100 mg/kg. Commercial samples of 2,4,5-T were used which were manufactured by Hercules Inc. and Dow Chemical. After repeating the Courtney-Moore study, Bionetics reported that only a small amount of fetuses were found to have developed abnormalities, less than in the reported HEW study of Courtney and Moore.[16] The Bionetics report implied that it was unlikely that the small number of developmental abnormalities detected was a sign of teratogenic action. However, on subsequent review it became clear than an error had occurred, but whether of reporting or of study design was never made clear. Actually, the dose used was 10 mg/kg of body weight and not 100 mg/kg of body weight as had originally been reported. With the discovery of that error, the observation of abnormalities obtained by that study now take on great significance. These are the lowest dose levels at which abnormalities have been reported for animals tested with 2,4,5-T containing .1 ppm of TCDD or less. Three percent of the fetuses examined in detail showed abnormalities. The developmental abnormalities in treated groups included cleft palate, umbilical hernia, renal agenesis, hydrocephaly, abnormal hind leg (possible club foot), heart defect, and epithelial defect.[17]

Finally there are the experiments by Roll[9] and by Neubert[10] mentioned before that obtained terata at doses of less than 40 mg/kg of technically pure 2,4,5-T.

Conclusion:

The data are by no means indecisive. Despite the poor experiments,

small number of animals used, and lack of adequate analysis, the weight of the evidence points to teratogenic effects of 2,4,5-T at much lower doses than 40 mg/kg of body weight.

Lethal and Chronic Health Effects of 2,4,5-T or TCDD

2,4,5-T is known to be toxic to animals.[18,19,20,21a,21b] There is considerable variation in 2,4,5-T toxicity and that must be kept in mind when generalizing about effects on humans. For instance, 2,4,5-T is lethal in small doses to dogs[19] but rats can tolerate relatively large doses. As has been pointed out by Sterling, one of the major design errors in many 2,4,5-T studies was the use of rats rather than of demonstrably more 2,4,5-T susceptible dogs or mice.[11]

TCDD even in minute quantities appears to be capable of causing chronic illness and death. One of the major concerns is the effect of TCDD on lymphoid tissue and its general impairment of an organism's basic defense system. TCDD in sublethal doses has been known to suppress the cell-mediated immunity in both mice and guinea pigs. Consistent with the observed suppression of the body's immune reaction are TCDD related lymphophenia in mice and guinea pigs.[22]

TCDD has been fed in minute quantities to various animals and found to be lethal.[23] In general, investigators who have had experience with trying to assess the toxicity of TCDD concluded that TCDD is the most potent porfirogenic chemical known.[24] Important is that the effects of TCDD appear to be cumulative.[25,26]

There is some suspicion that TCDD is mutagenic. While no mutagenic effect was reported in one in vitro study with bacteria exposed to 2,4,5-T,[27] two other studies did indeed report TCDD to be mutagenic.[28] In view of the studies involved, no definite conclusions can be drawn, but mutagenic effects are suspect. There is evidence of the carcinogenic potential of 2,4,5-T related TCDD. One experiment reported that intra peritoneal injections of 1 and 10 mg/kg of TCDD in the rat induced liver lesions.[29] The lesions appeared to be malignant on microscopic examination. The same investigator also reported a similarity between the effects of TCDD and known tetracarcinogenic compounds. Another investigator concluded that his experimental results pointed to the possibility that TCDD induces neoplasms.[30]

What appears clear from studies of TCDD's lethality is that because the effects of long-term exposures to low levels of TCDD remain undetermined, an acceptable level of exposure for neither man nor animal can be postulated. If TCDD exposure causes delayed lethality or if continued impingement of TCDD on human organs otherwise causes cumulative effects, or if TCDD concentrates in human tissue, a level of exposure which would be safe for the general population of either man or animal may not exist and even residues below the current level of detection may be unsafe.

B. THE POSSIBLE EFFECT OF 2,4,5-T ON MAN

It is true that physiological variations exist between animals that are tested to deduce the effect of toxic materials in man on whom such

tests cannot be performed. However, this difficulty in no way implies that man is unresponsive or less responsive to 2,4,5-T and TCDD than are test animals. Man may be more, rather than less, susceptible to some toxic materials, especially teratogens. The thalidomide experience is most important here. The lowest observed effective dose for human terata was .5 mg/kg/day. On the other hand, the hamster, dog, rat and mouse exhibited effects at 350, 100, 50 and 30 mg/kg/day respectively.[31] Thus, human mothers in terms of the lowest doses at which effects could be determined were some 60 times more sensitive than were mice. As a consequence, results of laboratory tests on mammalian species, especially mice, showing the teratogenic effects of 2,4,5-T present real grounds for concern.

This concern is intensified by the epidemiological information that has come out of Viet Nam. Because of the increased use of severe defoliation by the United States military and concern aroused by reports in Vietnamese newspapers in 1969, a survey was undertaken by the U.S. Army Medical Research Team.[32] The Cutting Report found no difference in still-birth and malformations between periods of relatively light and heavy defoliant spraying and no consistent differences between heavy and lightly defoliant-sprayed areas. However, there were a number of severe limitations to the army's study. Perhaps most damaging was that the populations most heavily exposed to 2,4,5-T were under-represented in the survey and inadequately dealt with in the records. The bulk of 2,4,5-T used in Viet Nam was sprayed in relatively remote and sparsely populated areas. The population directly exposed to 2,4,5-T probably did not exceed five percent and may have been one percent or less of the total population of Viet Nam. Very likely a significant proportion of the exposed population

consisted of Montagnards, whose births usually did not occur in hospitals. Consequently, a second survey was made by the Herbicide Assessment Commission (H.A.C.) of the American Association for the Advancement of Science.[33] The H.A.C. report by Meselson, Westing and Constable paid particular attention to records of the Tay Ninh Provincial Hospital, because although the total number of directly exposed Vietnamese is probably low, the northern portion of Tay Ninh had been heavily defoliated. The rivers draining the area of defoliation run through the remainder of the province and are a source of fish for some of the population. By examining records that were apparently not available to the Army survey, H.A.C. found that in 1968-69 the stillbirth rate recorded in Tay Ninh City Provincial Hospital was higher than rates found anywhere in the army survey. Also, by subtracting the capital area data and considering only the data from other parts of the country, H.A.C. showed that stillbirth and malformation rates increased from the years of light or no spraying to the years of heavy spraying. This would seem to indicate that in the remoter areas, where exposure could have been intense, stillbirth and malformation rates increased during the years in which spraying was heavy. Finally, H.A.C. noted an increase in the specific malformations of spinal bifida and cleft palate among admissions to the children's hospital. These malformations may be specifically related to 2,4,5-T exposure.

Another two studies, coming from North Viet Nam, document very carefully a number of cases of children with various malformations who were born to a relatively small sample of women who had been heavily exposed to 2,4,5-T defoliants, and a number of primary carcinomas of

the liver that are led back to the same source.[34]

Finally, there are a large number of anecdotal occurrences where reports of malformation of men and animals have been circulated but not verified. These include reports from Arizona and North West Washington in the United States, Sweden, and Finland, and British Columbia in Canada.[35]

A Remark on the Current Threshold-Dose Fallacy

Is it justified to extrapolate to a "clinical" threshold from a decreasing incidence of malformation as a function of dose? The answer is no.

Two types of dose-response curves can be obtained and each may relate to a different threshold. One dose response curve relates to the "intensity" of a reaction, for instance, graduated effects to different levels of poisoning. In such instances where a relationship exists between dose and level of reaction, it is possible to determine a safe dose, i.e. a sub-threshold dose beyond which no noticeable effect may be expected to happen.

Another dose-response relationship concerns the incidence with which an observable event happens as a function of dose. For instance, the incidence of lung cancer is related to the presence of irritating dusts and fumes containing micro-chemical carcinogens. The greater the concentration of these carcinogens, the greater will be the frequency or incidence of lung cancer and the smaller the dose, the smaller the relative frequency with which the disease will be observed. For very small doses, the probability of observing an incidence of disease may

become very small. But in no way is it possible to deduce a "safe" dose from such data. It is quite possible that any specific dose may be related both to frequency and intensity of disease. However, specific studies are needed to determine such relationships.

Investigations of the health effects of 2,4,5-T have established a relationship between dose and incidence of disease. There is nothing in the data that permits us to estimate a level of exposure below which the fetus is safe. Statements that claim that a pregnant woman needs to drink so and so much water or breath so and so much air contaminated with 2,4,5-T before causing a malformation are pure invention. They have no basis in any substantive evidence.

CONCLUSIONS

There seems to be no question but that TCDD is teratogenic, embryotoxic, fetogenic, may well be mutogenic and carcinogenic, and produces lethal and sublethal chronic health effects. There does not appear to be a safe dose of TCDD and probably not also of 2,4,5-T. It may well be that the so-called technically pure preparations of 2,4,5-T contain TCDD in parts per trillion rather than parts per million and that testing techniques have not been sensitive enough to measure the TCDD present in them. If this reasonable supposition is true, then all formulations of phenoxy herbicides that can form tetradyoxins are suspect of being potential teratogens and many have a number of other harmful effects.

Once TCDD has been introduced to the environment it appears to persist and to accumulate in the food chain. The discovery of TCDD in shrimp and crustaceans in Vietnam waters some four years after the cessation of defoliation is an exceedingly crucial discovery.[36] *The build up of this material in the food chain is implied in these findings and its impact on non-human and human ecologies may have severe consequences for man's survival. It is ominous therefore that the monitoring programs in the United States appear to find accumulation of TCDD in wildlife and cattle in the U.S. environment.*[37]

Frankly disturbing is the role played by industry scientists in the United States. We have given examples of data collected by industry that clearly showed the teratogenicity and toxicity of 2,4,5-T and TCDD in extremely small doses. Yet reports of experiments that produced these data did not draw attention to their findings. By not analyzing their data but presenting more or less undigested tabular listings of observations, these reports managed to mask observed effects that, at the very least, would have compelled further investigation. As a result, these reports give the false impression that 2,4,5-T or TCDD in small doses had no ontoward effect.

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