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ENVIRONMENTAL PROTECTION AGENCY

BEFORE THE HEARING EXAMINER

In re:	)	
	)	
2,4,5-Trichlorophenoxyacetic Acid (2,4,5-T)	)	I.F. & R. No. 295
	)	

PREHEARING BRIEF

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2,4,5-T Prehearing Brief

Table of Contents

I. Introduction

At the prehearing conference in this case on November 12, 1973, Judge Perlman requested that all parties submit a pretrial brief by January 18th, 1974. In support of our motion to intervene, the Environmental Defense Fund, Consumers Union, and Harrison Wellford (EDF, et al.) have already submitted a statement outlining in some detail our position on the issues in question. What follows, our prehearing brief, constitutes an elaboration upon that earlier submission, with the bibliography intended to double as our initial listing of proposed exhibits.

II. Human and Environmental Risks Posed by 2,4,5-T and Its Contaminants

Presence of TCDD in 2,4,5-T: Chlorodioxins, of which TCDD is one, are produced as contaminating by-products during the manufacture of 2,4,5-T and other chlorophenoxy compounds (1,2).*/ Therefore the adverse effects of 2,4,5-T cannot be assessed without taking into account those of TCDD, and the use of 2,4,5-T can be judged acceptable only insofar as the use of TCDD is acceptable. Although the proportion of dioxin contaminants in 2,4,5-T has been reduced following recent changes in manufacturing practice, we know of no evidence indicating that they have been or can be completely eliminated from commercial formulations of 2,4,5-T. In view of the extraordinary toxicity of TCDD, the massive amounts of 2,4,5-T which are sprayed, and the geographical scope of such spraying, even very low levels of TCDD in the herbicide formulations become unacceptable.

There is also the possibility that additional increments of dioxins may be formed in the environment when herbage treated with the 2,4,5-T mixture is burned. Whether such environmental formation of toxic dioxins actually occurs is apparently unknown at this time (3-5), adding a new dimension to the uncertainties concerning the safety of continued use of 2,4,5-T. Other chlorinated phenolic compounds may also occur as contaminants and may also be converted in the environment to toxic dioxins. Thus, trichlorophenol, 2 molecules of which can condense at high temperatures to form TCDD, occurs as a contaminant in 2,4,5-T (1).

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*/ References are listed by number in the attached bibliography, which also comprises our first list of proposed exhibits.

Toxicity: TCDD has been described as "perhaps the most potent small-molecule toxin known" (6) with an LD_{50} as a single oral dose for guinea pigs of 0.6 μg/kg of body weight (7). For growth inhibition, the no-effect level in guinea pigs has been reported as 8 x 0.04 μg/kg per week (8). In this same study, the lowest effective dose (growth inhibition) and the no-effect levels for rats were found to be, respectively 25 and 5 μg/kg (single dose) or 6 x 5 and 6 x 1 μg/kg/week. Toxicity to aquatic organisms is also extreme, with exposure of young salmon to more than 0.023 ppm for 24 hours having a delayed but irreversible lethal effect (9).

Human toxicity has also been shown. The presence of TCDD as a contaminant is believed to have been responsible for outbreaks of porphyria and chloracne among workers in plants manufacturing 2,4,5-T (10-13). The occurrence of porphyria, characterized by an increased urinary excretion of porphyins, is of particular interest in view of the demonstration that TCDD is a potent inducer of δ-amino-levulinic acid (ALA) synthetase in chicks (6) but not in rats (14). Because ALA is a precursor of porphyrins, the excessive porphyrin excretion in man, after exposure to TCDD, could be due to ALA synthetase induction, analogous to that which occurs in chicks. If the relative insensitivity of the rat to the toxic effects of TCDD (15,20) is related to the relative insensitivity to effects on enzyme induction, the human porphyria may indicate that the toxicity of TCDD in man more nearly resembles that in chicks than in the more resistant rat.

Because of contamination by TCDD, the exact degree of the toxicity of 2,4,5-T per se is uncertain but recent studies with relatively pure 2,4,5-T indicate that it too is toxic (see reviews in 16, 17). Thus, in the Bionetics study, a given maternal dose of 2,4,5-T containing 27-ppm TCDD produced a 30% incidence of cystic kidneys in the offspring. The same or higher dose of TCDD administered without the 2,4,5-T did not have this effect. This observation suggests either that 2,4,5-T itself caused the cystic kidneys or else that 2,4,5-T and TCDD have synergistic effects (see 17). FDA studies with chick embryos and hamsters show that terata and/or embryotoxicity were produced by an extensively purified sample of 2,4,5-T in which no dioxins were detectable (24).

Particularly ominous are the reports showing great differences in the sensitivities of different species to 2,4,5-T toxicity. The dog, for example, is more sensitive than the rat (18). Dogs are much closer to man, phylogenetically, than are rodents. Inferences about toxicity of 2,4,5-T based on experiments with laboratory rodents, therefore, may underestimate the degree of toxicity to man of 2,4,5-T and/or its contaminants (19). This possibility is reinforced by the observation that the monkey is more sensitive to a mixture of chlorinated dibenzodioxins (64% TCDD) than the rat (although less sensitive than the chicken (20)) and by the possibility, mentioned above, that humans may resemble chickens rather than rats in susceptibility to induction of ALA synthetase.

Teratogenicity: TCDD is a proven and potent teratogen, as are preparations of 2,4,5-T which contain TCDD (7, 21-23; see also 16, 17, 19 for citation of additional studies which have demonstrated teratogenicity). Few of the reported studies were designed in such a way as to be able to show convincingly a no-effect level of 2,4,5-T or TCDD. However, the dose required for teratogenesis is even lower than that for producing acute toxicity, being as little as 0.02 μg of TCDD per kg of embryonated egg for chick embryos (24). As with the toxicity studies, species differences in sensitivity to teratogenesis are also observed. In addition, synergism in the teratogenic effects of 2,4,5-T and of TCDD has been directly demonstrated (23).

Circumstantial indications that teratogenic effects in humans may have resulted from spraying of 2,4,5-T have been reported (25-28). Because large areas of land in Vietnam, as well as in the United States, have been sprayed with this known teratogen without any attempt to predict or monitor effects on the resident human and animal populations, these reports should not be denied or dismissed on the basis of lack of conclusive evidence (19).

Other adverse effects of 2,4,5-T and TCDD on animal reproduction (stillbirths, fetal deaths, embryotoxicity) are routinely observed experimentally. These experimental observations reportedly have their environmental counterparts in effects of spraying on domestic animals (16, 17, 29).

Mutagenicity: Mutagenic effects of 2,4,5-T and/or TCDD have been reported in several organisms. Very small doses of a commercial preparation of 2,4,5-T, containing less than 0.1 ppm TCDD, caused early oogenesis and chromosomal disturbances in Drosophila melanogaster (30); TCDD at concentrations of 2 and 4 ppm caused mutations in bacteria (31); and TCDD (0.02 or 1.0 ppb) produced a dramatic inhibition of mitosis, formation of dicentric chromosomal bridges and chromatin fusion in cells of the African blood lily (32). Although we are not aware of any reports of mutagenicity of these compounds in higher organisms, neither have we seen any reports of negative results in appropriate mutagenicity experiments.

Other adverse effects: Other miscellaneous pathological effects are produced in laboratory animals by TCDD and are described in more than a dozen papers published in Environmental Health Perspectives (Sept. 1973, #5). These additional effects include pre-natal and post-natal hydronephrosis, kidney and thyroid degeneration, growth inhibition, immunosuppression, thymic atrophy, leukopenia, thrombacytopenia, anemia, hepatic cellular necrosis, inhibition of biliary excretion, proliferation of hepatic smooth and rough endoplasmic reticulum, and induction of hepatic microsomal enzymes.

III Persistence, Mobility, and Accumulation of 2,4,5-T and TCDD in the Environment and in Food Chains

The fact that contaminant dioxins have the requisite toxicity and stability to cause serious adverse health effects when present in trace levels in food is convincingly demonstrated by the history of chick edema disease (33). An unknown dietary factor (CEF) caused millions of deaths in broiler flocks throughout a large part of the U. S. (in 1957 and 1959) and in England. Monkeys being used for experimental purposes were unexpectedly killed as well. Extracts of the flesh of chickens and hogs which had eaten the toxic rations were in turn found to be toxic, indicating that CEF is retained in an active form in the flesh of animals exposed to it. CEF was found to be present in commercially produced fatty acids including some of those destined for human consumption. Only after years of intensive research was the cause of this mysterious disease identified as the traces of dioxins present in feed, with TCDD being the most potent of the active compounds.

TCDD is chemically stable in soil (34) and is more soluble in lipids than in water. Compounds which possess these two characteristics can be expected to undergo bioconcentration and accumulation in animal food chains. Direct evidence that this does occur has been obtained experimentally (35-37). In one series of experiments (36) concentration factors for TCDD in aquatic food organisms ranged from 49 to 9222, depending on the experimental model used. The latter value was obtained under experimental

conditions analogous to those which presumably pertain in streams receiving runoff from previously sprayed fields, with TCDD being introduced as dried residue on sand particles. Under these conditions, TCDD concentration in a bottom-feeding food organism (mosquito larvae) was found to be 4,150 ppb (versus a water concentration of 0.45 ppb). Corresponding values for DDT were 12,000 and 2.40.

The presence of TCDD in human food, following spraying operations, has been demonstrated in a study of TCDD in fish and crustaceans sampled in Vietnam (38). TCDD was found in each of these Vietnamese samples in concentrations ranging from 0.02 to 0.81 ppb. The possibility that the TCDD had originated in the pentachlorophenol used as a wood preservative in Vietnam was ruled out (39). The samples were taken from six areas in Vietnam, all outside of but downstream from areas which had been heavily treated with 2,4,5-T. The stability of TCDD in the environment and in biological tissue is evidenced in this study not only by the fact that the compound was recovered in areas distant from the point of application, but also by the fact that the chemical analysis was not completed until 2-1/2 years after the samples were collected.

Capabilities in nature for microbial degradation of TCDD are rare. When 100 microbial strains with demonstrated ability to degrade persistent pesticides were tested, 5 strains produced slight degradation of TCDD whereas the others produced none (36).

An indirect suggestion of bioaccumulation is provided by studies with monkeys, rats, and guinea pigs which show that dioxin poisoning is cumulative and that repeated daily or weekly doses, each equaling only a small fraction of a "lethal" dose will also cause death (40, 8).

Analysis: With recent advances in methodology (38), it is now possible to measure dioxin in environmental samples containing extremely low concentrations. There should therefore be an absolute requirement that before further environmental releases are allowed, the distribution, persistance, and bioconcentration of TCDD resulting from past uses of 2,4,5-T should be thoroughly assessed. Such studies should also be required, perhaps in limited experimental ecosystems, with any new "uncontaminated" formulations of 2,4,5-T before their use is allowed. However, it must be remembered that failure to detect a toxic substance may merely reflect insufficiently sensitive methodology and equipment, despite the presence of the sought-after substance, and does not necessarily protect against its effects. As noted above, purified 2,4,5-T with no detectable dioxin was embryotoxic and teratogenic. Therefore, dioxin is effective at a concentration below the concentration detectable in 1970, or else 2,4,5-T is itself toxic and teratogenic even without dioxin. Either explanation is sufficient as a reason for terminating additional environmental releases of this herbicide.

In summary, commercial formulations of 2,4,5-T contain TCDD. It is apparent that these dioxins, being chemically and

biologically stable and fat soluble, may be retained in the environment and in animal tissues. The few observations which have been reported are in accord with this prediction. Because of the extraordinary toxicity and teratogenicity of TCDD, the unevaluated hazard of other contaminants, and the probable toxicity and teratogenicity of 2,4,5-T itself, the risks of continued use of 2,4,5-T are excessive.

IV. The Risks of 2,4,5-T's Continued Use Outweigh Any Possible Benefits

EDF, et al. do not oppose use of herbicides per se, and we wish to make this perfectly clear from the start. As we have already stated, however, we do oppose continued use of 2,4,5-T. While the applicable sections of FEPCA dealing with cancellation do not require proof of the existence of an alternative before a pesticide is cancelled, we believe that for every use of 2,4,5-T being defended in this hearing, there exist safe and effective chemical and non-chemical alternatives. Further, we contend that economic studies purporting to show the essentiality of continued use of 2,4,5-T (as opposed to all herbicides, for example), are grossly inadequate and distorted. Not only do we feel that such studies fail, inter alia, to take into account adverse effects of 2,4,5-T upon human health and the environment, but we further contend they fail entirely to consider the prevalence of patterns of misuse and unnecessary application. Given the fact that 2,4,5-T's method of application, aerial or ground spray, is inherently uncontrollable, we think that adverse effects are inevitable, and must be considered in any risk/benefit equation. We further think that such studies do not adequately consider the existence of alternatives, both chemical and mechanical, and thus greatly overstate the economic harm to users should 2,4,5-T be removed from the market. In addition, these studies fail to consider such factors as

economies of scale and attendant price reductions should sales of alternative products rise after 2,4,5-T is removed from the market.

V. Relief Sought

These hearings are an amalgam of FEPCA §§6B1 and 6B2 proceedings. Regarding the §6B1 portion of the hearing, we seek confirmation of the Administrator's already issued cancellation notice. As to the §6B2 portion of the hearing, we seek a final recommendation by the Administrative Law Judge that notices of cancellation be issues for all remaining non-cancelled registrations for 2,4,5-T.

We are also troubled by another question. While we are unclear about the extent of the Administrative Law Judge's authority in this regard, we further request that he at least consider recommending as part of his final opinion the stepwise reduction of residue tolerances of 2,4,5-T, its contaminants and metabolites, to zero in human foods. Whether or not we are completely successful, as we expect to be, in gaining all our cancellation relief sought, we think that the need to reduce residue tolerances to zero in human food, for 2,4,5-T, its contaminants and metabolites, will be made abundantly clear by the evidence to be presented in these hearings. We would

urge the Administrative Law Judge not to waste such an opportunity to recommend that remedial action be taken, even if he cannot himself order it.

Respectfully submitted,

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January 17, 1974

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I hereby certify that copies of the foregoing Prehearing Brief for EDF, et al. was mailed, first-class postage prepaid, to the following, this 18th day of January, 1974:

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