



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

AUG 17 1993

MEMORANDUM

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

SUBJECT: 107201. Fonofos (Dyfonate®). Corn Cluster Project.
Draft Toxicology Chapter in the Form of a RED

PC Code 041701
Tox. Chem. No. 454B

TO: Deborah L. McCall, Biologist
Special Review Section
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Pamela M. Hurley 8/12/93

THRU: Roger L. Gardner, Section Head
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Roger L. Gardner KB 8/12/93

Background and Request:

As part of the Corn Clusters Project, a risk assessment for Fonofos (Dyfonate®) is required. The Toxicology Branch (TB-I) has been requested to prepare the Toxicology Chapter (hazard assessment).

Toxicology Branch Response:

TB-I is submitting the Toxicology Chapter in the form of a Reregistration Eligibility Document (RED). Attached is a draft of the Chapter.

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FONOFOS: TOXICOLOGY CHAPTER FOR RED

Barcode No.

Case No.

Pesticide Chemical Code No. **041701**

CAS No.: 944-22-9

Submission No.

Tox. Chem. No. 454B

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ACUTE TOXICITY

The following summarizes acute toxicity values and categories for fonofos.

81-1	Acute Oral Toxicity in Rats Lab: Stauffer Chemical MRID 00078777 Report # T-6461 Date: 2/12/79 Acceptable	LD ₅₀ : 24.5 (21.4-28.0) mg/kg (males) LD ₅₀ : 10.8 mg/kg (9.6-12.2) (females) TOXICITY CATEGORY: I Tremors, salivation, diarrhea, lacrimation & labored breathing.
81-2	Acute Dermal Toxicity in Rabbits Lab: Stauffer Chemical MRID 00078777 Report # T-6461 Date: 2/12/79 Acceptable	LD ₅₀ : 159 (40-615) mg/kg TOXICITY CATEGORY: I Tremors, salivation, diarrhea, rapid breathing and miosis.
81-3	Acute Inhalation Toxicity in Rats MRID 419359-01 Lab: ICI Central Tox. Lab Report # HR2047 Date: 04/11/91 Acceptable	LC ₅₀ : 51.0 (33.5-77.7) µg/l (males) LC ₅₀ : 17.9 (8.6-37.0) µg/l (females) (Four hour exposure) TOXICITY CATEGORY: I Median lethal concentration was based on atmospheric concentrations. Clinical signs of toxicity and cholinesterase inhibition were evident and were consistent with the combination of neurological and irritancy effects which are typical of those seen following exposure to organophosphorus compounds.
81-4	Primary Eye Irritation in Rabbits Lab: Stauffer Chemical MRID: 00078777 Report # T-6461 Date: 2/12/79 Acceptable	Primary Irritation Score: Not given in DER. TOXICITY CATEGORY: IV Moderate irritation for 1/6 animals. 0.01 ml tested because in other studies with technical dyfonate, all animals died with a dose of 0.1 ml with no irritation. 0/6 died in this study.

81-5	<p>Primary Dermal Irritation in Rabbits Lab: Stauffer Chemical MRID: 00078777 Report # T-6461 Date: 2/12/79</p> <p>Acceptable</p>	<p>Primary Irritation Score: Not given.</p> <p>TOXICITY CATEGORY: IV 2/6 animals died. No irritation. 0.05 ml given as a dose. The required dose is 0.5 ml. However, all animals had died with a previous dose of 0.5 ml of the 93% Technical with Aliquot 335. The Tox. Category was I in the previous study.</p>
81-6	<p>Dermal Sensitization in Guinea Pigs Lab: ICI Central Tox. Laboratory MRID: 428426-01 Report # CTL/P/3195; Study #'s GG5133, GG5071 Date: 12/05/90</p> <p>Acceptable</p>	<p>Fonofos was tested for skin sensitization potential using a version of the maximisation test of Magnusson and Kligman. Formaldehyde was used as a positive control and elicited a positive response. Fonofos is considered to be a weak to mild sensitizer under the conditions of the study.</p>
81-8	<p>Acute mammalian neurotoxicity - rat Lab: Zeneca Central Tox. Labs, Alderley Park, UK MRID: 427778-01 Report # CTL/P/3946 Study # AR5434 Date: 3/17/93</p> <p>Core Grade:- Supplementary</p>	<p>NOEL: 4 mg/kg LOEL: 7 mg/kg</p> <p><u>Effects:</u> Dose levels: 0, 2, 4 or 7 mg/kg. Clinical signs of reduced foot withdrawal reflex, urinary incontinence, tip toe gait and upward curvature of the spine in 1 high dose female, which were totally reversible by 24 hours. Positive control data and results of preliminary range-finding study requested.</p>

SUBCHRONIC TOXICITY

No acceptable subchronic feeding studies are available in either the rodent or nonrodent. However, in the case of the rodent, an acceptable chronic/oncogenicity feeding study is available. For the nonrodent, a new chronic study has been requested. Two subchronic neurotoxicity studies are available, both of which are graded Core Supplementary, but may possibly be upgraded. The first study is a 90-day delayed neurotoxicity study in the hen (82-5; MRID No.: 401501-20) and the second study is a subchronic mammalian neurotoxicity study in the rat (82-7; MRID No.: 427926-01).

In the first study, technical Dyfonate^R was administered orally to adult hens for 90 days at 2, 4 and 8 mg/kg/day. Control groups were either untreated or given corn oil. The positive control group was administered tri-o-cresyl phosphate (TOCP). No evidence of delayed neurotoxicity was observed in any of the Dyfonate^R-treated hens, whereas the positive controls displayed marked evidence of delayed neurotoxicity in addition to progressive loss of body weight, inhibition of plasma cholinesterase, impaired egg production and death. The Dyfonate^R-treated animals exhibited significant weight loss in the high dose group, clinical signs of toxicity in the mid- and high dose groups (possibly the low dose group), inhibition of plasma cholinesterase in all dose groups and impaired egg production in all dose groups. The NOEL for inhibition of plasma cholinesterase is < 2.0 mg/kg/day (LDT) and the NOEL for other acute neurotoxic effects is < 2.0 mg/kg/day. The Agency has requested data from an acute delayed neurotoxicity study in the hen in order to conduct an overall assessment of the potential for Fonofos to induce delayed neurotoxicity. The 90-day neurotoxicity study in the hen has been classified as Core Supplementary until submission of data from the acute delayed neurotoxicity study. At that time, TB-I will reconsider the 90-day study for upgrading to an acceptable study for regulatory purposes. In addition, since this study is missing the assays for for acetylcholinesterase (AChE) and neuropathy target esterase (NTE) which are required under the new guidelines for hen studies, the regulatory requirement which this study will satisfy if upgraded, will apply to the previously published OPP neurotoxicity testing guidelines.

In the second study, Fonofos was tested in a 90-day feeding neurotoxicity screening battery in the rat at the following dose levels: 0, 15, 50 or 125/150 ppm in the diet (0, 0.75, 2.5, or 6.25/7.5 mg/kg/day). The NOEL is 2.5 mg/kg/day and the LEL is 6.25/7.5 mg/kg/day based on upward curvature of the spine, tiptoe gait, signs of urinary incontinence, pinched in sides, reduced splay reflex, splayed gait, eye bulging and shaking (females); increases in the mean time to tail flick (males), non-statistically significant increases in mean landing foot splay

(females) and in motor activity (statistically significant) at various times (females); significant and non-significant decreases in mean forelimb grip strength and in mean hindlimb grip strength (both sexes).

Neuropathological findings included a slight but statistically significant increase in mean brain weight in high dose females and 1 high dose female with a "collapsed brain, consistent with hydrocephalus". Microscopic findings included minimal nerve fibre degeneration in high dose males. These may be common to this strain of rat, but were considered in the determination of the NOEL.

The NOEL for cholinesterase inhibition is either at or near 0.75 mg/kg/day (LDT) based on statistically significant decreases in cholinesterase activity observed in both sexes for brain, erythrocyte and plasma at 2.5 mg/kg/day.

The other subchronic toxicity studies, 21- and 90-day dermal (82-2 and 82-3) and 90-day inhalation (82-4) studies are not required for the use patterns for Fonofos.

CHRONIC FEEDING

One acceptable chronic feeding study in rats is available (83-5; MRID No.: 406179-01). The study is classified as Core Minimum. A supplementary chronic feeding study in dogs has been used in the past for calculation of the RfD (83-1; MRID 000822-33). A new study has been requested in the FIFRA '88 process.

In the first study, Dyfonate was administered in the diet to Sprague-Dawley CD rats for 24 months at levels of 0, 4, 15, or 60 ppm and at 120 ppm for 12 months. The mean compound intake (averaged across sexes) was approximately 0.17, 0.65, 2.6 and 6.6 mg/kg/day at 4, 15, 60 or 120 ppm, respectively. Survival was not affected by dosing. The systemic NOEL is 2.6 mg/kg/day and the LEL is 6.6 mg/kg/day based on decreases in body weight and body weight gain. The NOEL for cholinesterase inhibition is 0.65 mg/kg/day and the LEL is 2.6 mg/kg/day based on inhibition of cholinesterase activity (brain, serum and erythrocyte). No effects of dosing were observed on other clinical laboratory findings or on organ weight, gross necropsy, or neoplastic findings.

In the second study, Dyfonate was tested in a chronic feeding study in dogs at 0, 16(8.0), 60 and 240 ppm for 2 years (0, 0.4(0.2), 1.5, 6 mg/kg/day). The cholinesterase NOEL is 0.2 mg/kg/day and the LOEL is 0.4 mg/kg/day (decreases in erythrocyte cholinesterase levels). The systemic NOEL is 0.4(0.2) mg/kg/day and the LOEL is 1.5 mg/kg/day [at 6 mg/kg/day: deaths, clinical signs, decrease in body weight, increase in serum alkaline phosphatase, possible liver effects (organ weights and

histopathology) and acute tissue congestion; at 1.5 mg/kg/day: a few clinical signs, liver weight increases and some possible body weight decreases, however, there were no major systemic effects]. This study is classified as Core Supplementary because the quality of the study was not sufficient for regulatory purposes. However, the study has been used in the past for calculation of the RfD, due to the low NOEL for cholinesterase inhibition. The major deficiencies included: an unusual feeding pattern; no information on the frequency of diet preparation, storage, stability of the test chemical in the diet, homogeneity of mixing or concentration analyses; in the high dose group, a replacement dog was started 6 weeks into the study and did not appear to be kept an extra 6 weeks at the other end of the study; no electrolytes were measured for the clinical chemistry analyses; the microscopic examinations were incomplete and statistical calculations were not conducted.

CARCINOGENICITY

Two studies are available: (1) chronic feeding/oncogenicity study in rats (83-5; MRID No.: 406179-01) and (2) oncogenicity study in mice (83-2; MRID No.: 401501-21). The mouse study is classified as Core Guideline and the rat study is classified as Core Minimum.

In the first study, Dyfonate was administered in the diet to Sprague-Dawley CD rats for 24 months at levels of 0, 4, 15, or 60 ppm and at 120 ppm for 12 months. The mean compound intake (averaged across sexes) was approximately 0.17, 0.65, 2.6 and 6.6 mg/kg/day at 4, 15, 60 or 120 ppm, respectively. Fonofos was not carcinogenic in this study.

In the second study, Dyfonate was administered in the diet to CD-1 mice for 18 months at levels of 0, 5, 25 or 100 ppm (males: 0, 1, 3, or 12 mg/kg/day; females: 0, 1, 4 and 15 mg/kg/day). It was not carcinogenic under the conditions of the study. The cholinesterase NOEL is 1 mg/kg/day and the LOEL is 3 mg/kg/day, based on inhibition of serum cholinesterase activity (males) and brain cholinesterase activity (males). At 12 mg/kg/day (15 for females), raised foci, masses, thickening, hyperplasia and hypertrophy of the duodenum (males); slight reductions in body weight, body weight gain and food consumption (males); reduction in serum cholinesterase (both sexes); reduction in brain cholinesterase (males) and inhibition of erythrocyte cholinesterase activity (both sexes) were observed. There were no significant effects on organ weights, hematology, ophthalmology, clinical signs and mortality.

DEVELOPMENTAL TOXICITY

Two studies are available: (1) developmental toxicity study in the rabbit (83-3a; MRID No.: 401501-22) and (2) developmental toxicity study in the mouse (83-3b; Accession No.: 248893 and MRID No.: 420576-01). Both studies are classified as Core Minimum.

In the first study, Dyfonate was tested in a rabbit developmental toxicity study at 0, 0.2, 0.5 or 1.5 mg/kg/day. The maternal NOEL is 1.5 mg/kg/day (HDT). The NOEL for developmental effects is also 1.5 mg/kg/day (HDT). This NOEL is borderline because there was a non-statistically significant increase in the number of resorptions/doe in the high-dose group. It was decided that this increase was not toxicologically significant because it was not statistically significant, it is within the historical control range and because the standard deviation for this measurement was so large.

In the second study, Dyfonate was tested in a mouse developmental toxicity study at 0, 2, 4, 6 or 8 mg/kg/day. The maternal NOEL is 6 mg/kg/day and the maternal LEL is 8 mg/kg/day, based on clinical signs of toxicity (tremors, chromodacryorrhea and dacryorrhea); decreases in body weight gain and slight decreases in food consumption. The NOEL for developmental effects is 2 mg/kg/day and the LEL is 4 mg/kg/day based on slight dilation of the 4th ventricle in the brain (4 mg/kg/day and above) and elevations in sternalbrae malalignment (6 mg/kg/day and above).

REPRODUCTION

No acceptable studies are available.

MUTAGENICITY

Three studies are available: (1) gene mutation assay in S. typhimurium (84-2a; MRID No.: 417692-01); (2) in vitro cytogenetics assay in human lymphocytes (84-2b; MRID No.: 418371-01); and (3) in vivo micronucleus assay in mice (84-2c; MRID No.: 418133-01). All three studies were negative and were classified as Acceptable.

In the first study, Fonofos was tested for potential to induce reverse mutations in Salmonella typhimurium, both with and without metabolic activation at the following dose levels: 0.32, 1.6, 8.0, 40, 200, 1000 and 5000 ug/plate. Fonofos was tested up to levels of cytotoxicity. It did not induce a significant increase in the number of reverse mutations when compared to the vehicle control, DMSO and to the absolute control.

In the second study, Fonofos was tested for potential to induce chromosomal aberrations in an in vitro assay in human lymphocytes up to cytotoxic levels. The dose levels tested were 10, 50 and 100 ug/ml both with and without metabolic activation. Fonofos did not induce a significant increase in chromosomal aberrations under the conditions of the study. Positive controls verified the sensitivity of the assay.

In the third study, Fonofos was tested in a mouse micronucleus test at 6 and 9.5 mg/kg. There were no statistically or biologically significant increases in the frequency of micronucleated polychromatic erythrocytes in mice treated with fonofos at either dose level at any of the sampling times investigated, when the data from both sexes were considered separately or when combined (when compared to vehicle control values). The percentage of polychromatic erythrocytes in the treated animals when compared to the controls indicates that there was some indication of cytotoxicity to the bone marrow cells at the dose levels tested. , !

METABOLISM

Seven metabolism studies are available (85-1; MRID Nos. 00090876, 00043508, 00090875, 00090877, 00090879, 00090800, 00092025). The first study was classified as Acceptable and the rest of the studies were classified as Core Supplementary. However, all of the studies when taken together satisfy the regulatory requirements for metabolism studies.

Single oral dose studies in male rats indicated that by 48 hours, greater than 94% of radioactive doses (C^{14} in the ethyl moiety) were eliminated in the urine and feces. In an oral study, 87.6% and 13.7% of dyfonate was eliminated as identified metabolites in urine and feces, respectively. In the urine, o-ethylethanephosphonothioic acid, o-ethylethanephosphonic acid (EOP), methylphenylsulfone and its phenyl hydrolates have been identified as metabolites. In the feces, methylphenylsulfone and EOP have been identified as metabolites. Using a study in cannulated rats, it was calculated that there was approximately 15% enterhepatic recirculation of dyfonate. Excretion of a low dose of dyfonate administered orally over 16 days was not affected by previously induction of hepatic enzymes with dyfonate. In single oral dose studies with the metabolite, thiophenol in both sexes, at 60 hours the radiolabel was excreted at 94.4% and 75.8% in urine and feces combined for high and low dose, respectively. Two percent was found in tissues, mostly hair and hide.

OTHER TOXICOLOGICAL ENDPOINTS

A dermal penetration study (85-2) with technical grade fonofos is not required at this time because there are no toxicological endpoints to indicate that this study is required.

Domestic animal safety studies (86-1) are not required for the use patterns of fonofos (a soil insecticide for corn borers and rootworms, cutworms, symphylans, wireworms and other soil and foliar pests).

REFERENCE DOSE

On August 12, 1993, the HED Reference Dose (RfD) Peer Review Committee recommended that the RfD for fonofos be established at 0.007 mg/kg/day. This value was based on co-critical studies: the rat cholinesterase NOEL of 0.65 mg/kg/day from the rat chronic feeding/oncogenicity study (83-5; MRID No.: 406179-01) and on the rat cholinesterase NOEL of 0.75 mg/kg/day from the rat subchronic mammalian neurotoxicity study (82-7; MRID No.: 427926-01) and an uncertainty factor (UF) of 100. This RfD has not yet been confirmed by the Agency RfD Work Group.

BIBLIOGRAPHY

<u>Guideline Number</u>	<u>MRID Number</u>	<u>CITATION</u> *
81-1	00078777	Holmes, P.A. (1978?) Dyfonate Technical: T-6461. (Unpublished study received May 7, 1981 under 476-2028; submitted by Stauffer Chemical Co., Richmond, Calif.; CDL:245491-A)
81-2	00078777	Holmes, P.A. (1978?) Dyfonate Technical: T-6461. (Unpublished study received May 7, 1981 under 476-2028; submitted by Stauffer Chemical Co., Richmond, Calif.; CDL:245491-A)
81-3	419359-01	Lewis, R.; Mould, A. (1991) Fonofos: 4-Hour Acute Inhalation Toxicity Study in the Rat: Lab Project Number: CTL P 3307: HR2047. Unpublished study prepared by ICI, Alderly Park. 169 p.
81-4	00078777	Holmes, P.A. (1978?) Dyfonate Technical: T-6461. (Unpublished study received May 7, 1981 under 476-2028; submitted by Stauffer Chemical Co., Richmond, Calif.; CDL:245491-A)
81-5	00078777	Holmes, P.A. (1978?) Dyfonate Technical: T-6461. (Unpublished study received May 7, 1981 under 476-2028; submitted by Stauffer Chemical Co., Richmond, Calif.; CDL:245491-A)
81-6	42842601	Rattray, N.; Robinson, P. (1990) Fonofos: Skin Sensitisation to the Guinea Pig: Lab Project Number: CTL/P/3195: GG5133: GG5071. Unpublished study prepared by ICI Central Toxicology Lab. 30 p.
81-8	427778-01	Horner, H. (1993) Fonofos: Acute Neurotoxicity Study in Rats: Lab Project Number: CTL/P/3946: AR5434. Unpublished study prepared by Zeneca Central Toxicology Lab. 318 p.
82-5	40150120	Miller, J. (1987) Neurotoxicity of 90-day Oral Administration of Technical Dyfonate to Adult Hens: T-6237: Final Report. Unpublished study prepared by Stauffer Chemical Co. 59 p.

<u>Guideline Number</u>	<u>MRID Number</u>	<u>CITATION</u> *
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		Pulsford, A. (1991) First Amendment to a Teratology Study in CD-1 Mice with Dyfonate Technical (MRID No. 118423): Lab Project No. T-10192, T-10192C. Unpublished study prepared by Stauffer Chemical Co. 8 p.
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84-2a	41769201	Callander, R. (1990) Fonofos: An Evaluation of Mutagenic Potential Using S. Typhimurium: Lab Project Number: CTL/P/3153: YV2906. Unpublished study prepared by ICI Central Toxicology Laboratory. 34 p.
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85-1	00090875	Hoffman, L.J.; Ford, I.M.; Menn, J.J. (1971) Dyfonate metabolism studies: 1. Absorption, distribution and excretion of O-ethyl S-phenyl ethylphosphonodithioate in rats. Pesticide Biochemistry and Physiology 1(3-4):349-355. (Also In unpublished submission received Dec 13, 1977 under 476-1995; submitted by Stauffer Chemical Co., Richmond, Calif.; CDL:232472-F)

<u>Guideline Number</u>	<u>MRID Number</u>	<u>CITATION</u> *
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*Each study listed in this bibliography was conducted with technical grade Fonofos (Dyfonate).