APPENDIX F. Ecological effects studies considered

MRID	Taxa		AI	Study Time	Endpoint	Toxicity	NOEL	Date	Classification
42836103	Aquatic Plant	Marine diatom *Skeletonema costatum*	98.4	5 D	NOAEC	<11.0 ppm	N.R.	1996	Supplemental
42836101	Aquatic Plant	Duckweed *Lemna gibba*	98.4	14 D	NOAEC	<11.0 ppm	N.R.	1996	Supplemental
42882401	Aquatic Plant	Freshwater diatom *Navicula pelliculosa*	98.4	5 D	NOAEC	<11.0 ppm	N.R.	1996	Supplemental
42836102	Aquatic Plant	Bluegreen algae *Anabaena flos-aquae*	98.4	5 D	NOAEC	<11.0 ppm	N.R.	1996	Supplemental
41054829	Aquatic Plant	Green algae *Scenedesmus subspicatus*	96.7	96 hr	EC50	>solubility	N.R.	1990	Acceptable
41155714	Aquatic Plant	Green algae *Selenastrum capricornutum*	98	7days	EC50	>solubility	3.09	1990	Acceptable

MRID	Taxa	Common Name	Age	Guideline	Type	AI	Study Time	Toxicity	Date	Classification
41155706	Aves	Bobwhite quail *Colinus virginianus*	10 D	71-2	D	100	8 D	LC50>5620 ppm	1989	Acceptable
41155705	Aves	Bobwhite quail *Colinus virginianus*	27 WKS	71-1	O	100	14 D	LC50>2250 mg/kg	1989	Acceptable
41155707	Aves	Mallard duck *Anas platyrhynchos*	Juv	71-2	D	100	8 D	LC50>5620 ppm	1989	Acceptable

MRID	Taxa	Common Name	Age	Guideline	Type	AI	Study Time	Endpoint	Toxicity	Units	CL	Date	Classification
TN 0933, 40226901	Crustacea	Water flea *Daphnia magna*	<24 hr	72-2	S	75	48 hr	EC50	>solubility	PPM	98-193	1976	Supplemental
40098001	Crustacea	Water flea *Daphnia magna*	1st-I	72-2a	S	100	48 hr	EC50	>solubility	PPM	N.R.	1986	Supplemental
40098001	Crustacea	Water flea *Daphnia magna*	1st-I	72-2b	S	75	48 hr	EC50	>solubility	PPM	20-35	1986	Supplemental
40098001	Crustacea	Scud *Gammarus pseudolimnaeus*	Mat	72-2b	S	75	96 hr	LC50	>solubility	PPM	3.4-11.3	1986	Supplemental
40228401	Crustacea	Brown shrimp *Penaeus aztecus*	Juv	72-3c	F	100	48 hr	LC50	>solubility	PPM	N.R.	1986	Supplemental
40098001	Crustacea	Scud *Gammarus pseudolimnaeus*	Mat	72-2a	S	100	96 hr	LC50	>solubility	PPM	N.R.	1986	Supplemental

MRID	Taxa	Common Name	Age	Guideline	Type	AI	Study Time	Endpoint	Toxicity	Units	CL	NOEL	Date	Classification
TN 0908, 40227001	Fishes	Rainbow trout *Oncorhynchus mykiss*	0.33 g	72-1	S	75	96 hr	LC50	>solubility	PPM	22.14-40.64	N.R.	1987	Supplemental
41054826	Fishes	Rainbow trout *Oncorhynchus mykiss*	N.R.	72-1	S	96.7	96 hr	LC50	>solubility	PPM	N.R.	N.R.	1990	Supplemental
TN 0878, 40227002	Fishes	Bluegill sunfish *Lepomis macrochirus*	0.54 g	72-1	S	75	96 hr	LC50	>solubilty	PPM	21-180	120	1975	Supplemental
41054827	Fishes	Bluegill sunfish *Lepomis macrochirus*	1.27 g	72-1	S	96.7	96 hr	LC50	>solubility	PPM	N.R.	N.R.	1990	Supplemental

40228401	Fishes	Sheepshead minnow *Cyprinodon variegatus*	Juv	72-3a	F	100	48 hr	LC50	>solubility	PPM	N.R.	N.R.	1986	Supplemental
40098001	Fishes	Bluegill sunfish *Lepomis macrochirus*	0.6 g	72-1	S	75WP	96 hr	LC50	>solubility	PPM	N.R.	N.R.	1986	Supplemental
TN 0437	Fishes	Bluegill sunfish *Lepomis macrochirus*	0.41 g	72-1	S	2.5	96 hr	LC50	>solubility	PPM	NA	100	1972	Supplemental
TN 0481/00107142	Fishes	Rainbow trout *Oncorhynchus mykiss*	0.873	72-1	S	2.5	96 hr	LC50	>solubility	PPM	N.R.	180	1987	Supplemental

MRID	Taxa	Common Name	Age	Guideline	Type	AI	Study Time	Toxicity	Units	CL	NOEL	Date	Classification
40098001	Insecta	Midge *Chironomus plumosus*	3rd-I	72-2a	S	100	96 hr	LC50>100	PPM	N.R.	N.R.	1986	Supplemental

MRID	Taxa	Common Name	Age	Guideline	Type	AI	Study Time	Endpoint	Toxicity	Date	Classification
41564901	Terrestrial Plant	Tomato *Lycopersicon esculentum*	Seedlg	123-1a	SErw	97.8	14 D	EC25	Not Determined	1992	Supplemental
41440101	Terrestrial Plant	Soybean *Glycine max*	JuvPln	123-1b	VVrw	Tech	14 D	EC25	Not Determined	1992	Supplemental

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

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

July 8, 2002

MEMORANDUM:

SUBJECT: Human Health Risk Assessment for Chlorthal dimethyl (DCPA) to Support New Uses on California Parsley and Other Minor Crops. PP#s 0E3883 and #2E6442. Registration Case No. 194122. Chemical No. 078701. DP Barcode D281320.

FROM: Timothy C. Dole, CIH, Industrial Hygienist
William J. Hazel, Ph.D., Chemist
Kit Farwell, D.V.M., Toxicologist
Reregistration Branch 1
Health Effects Division (7509C)

THRU: Whang Phang, Branch Senior Scientist
Reregistration Branch 1
Health Effects Division (7509C)

TO: Robert Forrest, Chief
Minor Use, Inerts and Emergency Response Branch
Registration Division (7505C)

Please find attached the Human Health Risk Assessment to support the use of chlorthal dimethyl (DCPA) on minor crops to include basil, celeriac, chicory, chives, coriander, dill, ginseng, marjoram, oriental radish, radicchio and parsley. This assessment is based upon the Toxicology Chapter (TXR 0050574), the Dietary Exposure Assessment Memorandum (D283274) and the Occupational and Residential Exposure Assessment (D283509). Information was also drawn from the EFED's Drinking Water Assessment of 5/06/2002 and the FQPA Safety Factor Committee memorandum of 5/22/2002. This risk assessment or its components have been evaluated within HED by the following peer review committees: HIARC, FQPA SFC, ChemSAC, ExpoSAC, DE SAC, and RARC, and it includes the comments and recommendations of these committees.

Table of Contents

DCPA

HED'S HUMAN HEALTH RISK ASSESSMENT

1.0 EXECUTIVE SUMMARY

Overall Summary

The Health Effects Division (HED) has conducted a human health risk assessment for the active ingredient **DCPA** for an IR4 petition for proposed new uses on minor crops such as parsley and ginseng. HED determined that the data are adequate to support the petition. The occupational risks are not of concern. The food and residential handler non-cancer and cancer risks are not of concern either separately or when aggregated. The food and residential post application turf cancer risks are not of concern when aggregated if the turf is irrigated after application. The DWLOC values are less than the EECs for both chronic non-cancer risks and for cancer risks.

Introduction

DCPA [dimethyl tetrachloroterephthalate] is a pre-emergence phthalic acid herbicide registered for use on a variety of vegetables, strawberries and turf. The proposed new uses include basil, celeriac, chicory, chives, coriander, dill, ginseng, marjoram, oriental radish, radicchio and parsley. DCPA will be applied at a rate of 4.5 to 10.5 lb ai per acre to the soil prior to crop seed germination. DCPA is classified as a photosynthesis inhibitor and plant growth regulator. A RED was completed for DCPA in 1998 and was based upon a risk assessment completed by HED in 1995. The RED concluded that all of the uses except turf were eligible for reregistration. The turf uses were of concern because of the cancer risk of DCPA and of hexachlorobenzene (HCB) which is a manufacturing impurity. The original supplier (ISK Biosciences) stopped producing DCPA in 1997 and a new supplier (AMVAC) started production in 2000. The labels for the technical product and for Dacthal W-75 were approved by RD in 2001. The other new labels from AMVAC are currently being reviewed by SRRD.

Product chemistry data submitted by AMVAC indicates that the AMVAC DCPA technical has an HCB upper certified limit of 40 ppm. According to the 1998 RED, the maximum allowable HCB impurity level was 0.3 percent (3000 PPM).

Toxicology

<u>Non-Cancer Effects</u>

The acute toxicity of DCPA is low (Tox III and IV). It is a mild irritant to the eyes and skin and it is not a skin sensitizer. The major effects seen during the toxicity studies include decreased thyroxine levels in parallel with thyroid histological changes and liver hypertrophy. It is believed that the liver effects are precursor to the thyroid effects.

There were no significant sex differences in the metabolism of radio-labeled DCPA. Absorption was more efficient at 1 mg/kg/day (79%-86% of administered dose) than at 1000 mg/kg/day (6-9%). DCPA was excreted in the urine primarily as the mono-acid metabolite. Less than 1% was found in bile, so DCPA in feces represents unabsorbed compound. Although 12% of the administered dose was found in fat 12 hours after discontinuance of dosing , the dose had depleted to 0.03% by 168 hours. Concentration of DCPA in the thyroid reached a maximum at 36 hours and rapidly depleted by 168 hours. By 168 hours, highest concentration of DCPA was found in the kidney.

Liver enzymes were elevated in the chronic mouse and rat studies. Thyroid toxicity in the chronic rat study consisted of decreased levels of thyroxine, follicular cell hyperplasia/hypertrophy, and increased thyroid weight. Thyroid follicular cell hyperplasia or hypertrophy occurred in the subchronic rat feeding studies and in the reproduction study. Thyroid hormones were not evaluated in the subchronic rat studies. Thyroid histological changes were not seen in the chronic mouse study.

The data from the developmental studies in rats and rabbits indicated that there are no concerns for either quantitative or qualitative susceptibility. The data from a 2 generation reproductive study in rats indicated no quantitative or qualitative susceptibility because the offspring No Observed Adverse Effect Level (NOAEL) is the same as the parental NOAEL. The parental effects, especially renal and thyroid toxicity, were more severe than the reversible effects upon pup body weight.

Kidney weight was increased in the 90-day and chronic rat studies. There were increased incidences of chronic nephropathy in the chronic rat study and the reproduction study, along with increases in BUN and creatinine in the chronic rat study. There was anemia in the chronic rat study. An increased incidence of pneumonitis, diagnosed by histopathology, was seen in the chronic rat study and the reproduction study.

The following mutagenicity studies were negative: two Mouse lymphoma assays, a cytogenetic assay in Chinese Hamster Ovary (CHO) cells, two unscheduled DNA synthesis assays, and an assay for sister chromatid exchange in CHO cells. The Ames test study was classified as unacceptable.

Cancer Effects

Thyroid follicular cell adenomas were increased in male and female rats exposed to DCPA during a 104 week dietary study while thyroid follicular cell carcinomas were increased only in female rats. Hepatocellular adenomas, hepatocellular carcinomas and hepatocholangio-carcinomas were increased only in female rats. In a mouse dietary study, hepatic adenomas, but not carcinomas, were increased in females. Hepatic adenomas and carcinomas were also observed in males.

Hepatocellular carcinomas were observed in female rats during a 104 week dietary study of HCB.

Dose Response and Endpoint Selection

The HIARC concluded that the toxicology database for DCPA is not complete, however, it was adequate for selecting toxicity endpoints for risk assessment. The main uncertainty is extrapolation of the thyroid effects observed during the chronic study to short and intermediate exposure durations because thyroid hormones were not measured during the 28-day dermal and 90-day feeding studies. No doses were established for acute dietary exposures because no appropriate endpoint from a single exposure was identified in any of the toxicity studies. No doses were established for short/intermediate term dermal exposures because no systemic toxicity occurred in the dermal toxicity study at the high dose of 1000 mg/kg/day. The following endpoints were used in this risk assessment and were assessed using uncertainty factors of 10X for interspecies extrapolation and 10X and intraspecies variability.

- Chronic Dietary, NOAEL = 1 mg/kg/day based on decreased thyroxine levels and liver and thyroid histological changes in male rates with a LOAEL of 10mg/kg/day.

- Incidental Oral (short and intermediate term), NOAEL = 50 mg/kg/day based on increased incidence of hepatocellular hypertrophy in a 90-day feeding study in rats with a LOAEL of 100 mg/kg/day.

- Inhalation (Short and Intermediate Term), NOAEL = 50 mg/kg/day based on increased incidence of hepatocellular hypertrophy in a 90-day feeding study in rats with a LOAEL of 100 mg/kg/day.

The DCPA rat dietary study was used to determine a Q1* of 0.0015 $(mg/kg/day)^{-1}$ for DCPA and the HCB rat dietary study was used to determine a Q_1^* of 1.0 $(mg/kg/day)^{-1}$ for HCB.

FQPA Considerations

The FQPA Safety Factor is 1X based upon the available hazard and exposure data and is applicable to all population subgroups and exposure scenarios. There was no evidence of pre- or post-natal susceptibility from *in utero* or postnatal exposure to DCPA. There are no residual uncertainties.

Dietary Risk

The DCPA dietary risk analyses reflect largely refined exposure assessments for the existing crop uses. Anticipated residues (ARs) and percent crop treated information were incorporated. Tolerance level residues and 100 percent crop treated were used for the proposed crop uses. ARs were calculated using mainly field trial data. In the case of lettuce, FDA monitoring data combined with crop rotational field trial data were used.

Chronic dietary risks calculated using a chronic PAD of 0.01 mg/kg/day were low (<2% cPAD) for all population subgroups of concern. Cancer risks were also not of concern with an estimated lifetime risk to the general population of 1.5×10^{-7}.

Drinking Water Risks

The EFED provided the drinking water assessment using simulation models to estimate the potential concentration of DCPA and the metabolite TPA in surface and ground water. Parent DCPA is not especially persistent or mobile and substantial amounts could be available for runoff for several weeks post-application. Most of this runoff will generally occur in the form of adsorption to eroding soil, however, as opposed to dissolution in runoff water. TPA appears to be substantially more persistent than parent DCPA and exhibits low soil/water partitioning. Therefore, substantial quantities of TPA should be available for runoff for a longer period than the parent DCPA.

PRZM-EXAMS modeling was done for DCPA and TPA assuming assuming application of DCPA to turf at the maximum label rate of 15 lbs ai/acre with a percent cropped area (PCA) of 87 percent or assuming application of DCPA to cotton at the maximum label rate of 10.5 lbs ai/acre with a PCA of 20 percent. The estimated environmental concentration (EEC) of DCPA in surface water is 42 or 11 ug/liter for the annual mean concentration and 33.1 or 8.0 ug/liter for the 36 year mean concentration. The EEC for TPA in surface water is 321 or 366 ug/liter for the annual mean concentration and 160 or 220 ug/liter for the 36 year mean concentration. SCI-GROW modeling was done to estimate the 90 average ground water concentrations for turf and cotton using the same application rates and PCAs as for the surface water modeling. The EECs were 0.014 or 0.0099 ug/liter for DCPA and 275 or 192 ug/liter for TPA.

There are limited surface and ground water monitoring data available for DCPA and TPA and these data are not adequate to perform a quantitative drinking water assessment.

Residential Handler and Post Application Risks

The inhalation exposures of residential handlers mixing/loading and applying DCPA to lawns and gardens were assessed using maximum label rates and standard SOP assumptions. The MOEs were all above the target of 100 and are not of concern. The cancer risks for the residential handler scenarios were calculated assuming two applications per year. Both the dermal and inhalation routes of exposure were added to determine the lifetime average daily dose. None of the scenarios are of concern for either DCPA or HCB because the cancer risks are below 1.0×10^{-6}.

Post application exposures are not anticipated for garden vegetables because the applications are made to freshly cultivated soil using only the granular products.

Post application exposures are anticipated for turf because broadcast applications are made to prevent the growth of weeds throughout the lawn. These exposures are anticipated to be short term because only one or two applications are made per growing season and the label recommended application interval is two months or longer. A Turf Transferable Residue study had been submitted in support of the RED and this study generally complied with series 875 guidelines. The study data indicated that irrigation reduced the DAT 0 residue by 62%.

The cancer risks for adults exposed to treated turf were calculated using the TTR data averaged over 14 days and assuming four days per year of exposure to turf treated within 14 days. The cancer risks for adults performing yard work on treated and irrigated turf was 3.4×10^{-7} for

DCPA and 1.1 $x10^{-8}$ for HCB. The DCPA and HCB cancer risks for adults playing golf were 29 times less than the risks for yard work and are not of concern.

Standard assumptions from the Draft Standard Operating Procedures for Residential Exposure Assessment and updated input values from the Recommended Revisions of 22 February 2001 were used to calculate incidental oral exposure for toddlers exposed to treated turf. All of the MOEs exceeded the target of 100 on day after treatment zero (DAT 0), and are not of concern.

Aggregate Risks and Drinking Water Levels of Concern

The exposures of all the co-occurring pathways were aggregated and compared to the appropriate endpoint to determine the drinking water level of concern (DWLOC) . These pathways include dietary food exposure, residential handler exposure and residential post application exposure. Inhalation exposures were considered for residential handler scenarios while only incidental oral exposures were considered for toddler post application exposure. An acute endpoint was not identified by the HIARC; therefore, no acute aggregate risk assessment is required. The short/intermediate DWLOCs are greater than the combined estimated water concentrations (EEC) of DCPA and TPA and are not of concern. Since no chronic residential scenarios have been identified, chronic DWLOCs for DCPA were calculated based on anticipated residues in food alone. These DWLOCs are less than the combined EEC which indicates that the drinking water risk may be of concern for chronic exposures. It should be noted that the aggregate chronic risk excluding drinking water is low ($\leq$1.1% of the cPAD).

Cancer DWLOCs for DCPA were calculated using food alone and together with the residential handler or heavy yard work exposure scenarios. These DWLOCs were 3.5 to 5.1 times less than the corresponding EECs, which indicates that the drinking water exposures may be of concern. The EECs for the metabolite TPA are much higher than the EECs for DCPA and are the risk drivers. The aggregate cancer risks excluding drinking water ranged from $1.5x10^{-8}$ when food alone is considered alone to 6.8×10^{-7} when food and residential handler exposures are considered.

Occupational Risks

Workers may experience short term exposures to DCPA during mixing, loading and application. Dermal exposures were not assessed for non-cancer risks because dermal toxicity testing indicated that the no effects were observed at the highest dose tested (1000 mg/kg/day). Dermal exposures were assessed for cancer risks using the dermal absorption factors of 22% for DCPA and 27% for HCB. HCB exposures were calculated assuming that DCPA contains 40 ppm HCB by weight as a manufacturing impurity. The MOEs for non-cancer risk are all greater than 100 if label required PPE is used and are not of concern. The cancer risks were assessed for DCPA and HCB assuming one exposure day per year for application to minor crop fields. The cancer risks for DCPA using label required PPE range from 2.6×10^{-7} to $1.1x10^{-6}$ for mixing and loading and are $8.0x10^{-8}$ for application. The cancer risks for HCB are 30 times less than the cancer risk for DCPA and are not of concern.

Risk Characterization and Data Requirements

Although the DCPA databases were substantially complete, confidence in several areas of the risk assessment would improve with the following data:

- A guideline 28-day inhalation study in rats, which must include assessment of thyroid histopathology and thyroid hormone levels.

- A confirmatory study showing the comparative short-term thyroid toxicity of DCPA in adults and offspring. This study should include evaluation of thyroid hormone levels and liver induction. The registrant should consult with the Agency to discuss this study before beginning the study.

The following information could be used to refine the risk estimates.

- Actual use rates and percent crop treated for DCPA applied to residential turf and golf courses. The highest label rate of 15 lbs ai/acre was used by EFED in the drinking water assessment. This rate is only applied in the fall for annual blue grass control. It is not known if the typical homeowner would attempt to control annual bluegrass since it is similar in appearance to desirable grass species. It is more likely that the homeowner would focus on crabgrass control which uses a lower rate of 10.5 lb ai/acre. Golf course managers would probably be interested in controlling both crabgrass and annual bluegrass, however, their use of DCPA is limited by the fact that DCPA cannot be used on putting greens.

- Actual use rates and percent cropped treated for cotton. The QUA report indicated that there was no reported use of DCPA on cotton for the years 1995-2000. NASS data for 2001 indicated that DCPA was applied to less than 1% of the cotton crop in 2001.

Tolerance Reassessment

A listing of the new tolerances that were established for the minor crops is included in Table 1. All of the existing DCPA tolerances established at 40 CFR 180.185 are also considered by HED to be reassessed. Please refer to the dietary exposure and risk assessment prepared by W. Hazel (6/5/02; D283274).

Table 1. Tolerance Summary for Combined Residues of DCPA, MTP, and TPA			
Commodity (as proposed)	Proposed Tolerance (ppm)	Correct commodity definition[a]	Recommended Tolerance (ppm)[a]
Oriental radish	2	Radish, oriental	2
Basil	5	Basil, **fresh leaves**	5
--	--	**Basil, dried leaves**	**20**
Coriander	5	Coriander, **leaves**	5
Dill	5	Dill	5
Marjoram	5	Marjoram	5
Chives	5	Chive	5
Ginseng	2	Ginseng	2
Celeriac	2	Celeriac	2
Chicory	5	**Chicory, roots**	**2**
--	--	Chicory, **tops**	5
Radicchio	2	Radicchio	**5**
Parsley (Fresh)	5	Parsley, **leaves**	5
Parsley (Dried)	15	Parsley, **dried leaves**	**20**

[a]Corrections/recommendations in bold.

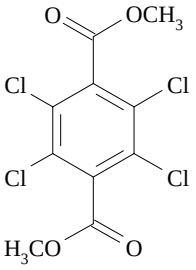
2.0 PHYSICAL/CHEMICAL PROPERTIES CHARACTERIZATION

Chemical Structure and Identification

DCPA [dimethyl tetrachloroterephthalate] is a pre-emergence phthalic acid herbicide registered for use on a variety of vegetables, strawberries and turf. It is classified as a photosynthesis inhibitor and plant growth regulator.

According to a search of the Reference Files System (REFS) conducted 5/03/02, there is one registered manufacturing-use product under PC Code 078701, AMVAC Chemical Corporation's 98.9% technical (EPA Reg. No. 5481-495). This technical was produced by ISK Biosciences until 1997, after which production was discontinued until AMVAC started production in 2001.

The chemical structure of DCPA is shown below:



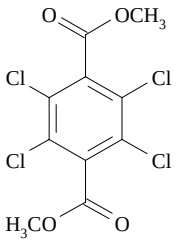
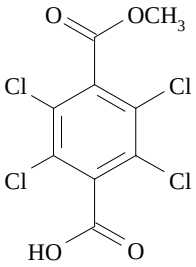
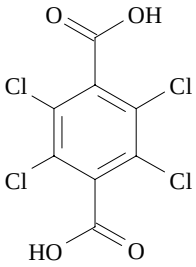
Empirical Formula:	$C_{10}H_6Cl_4O_4$
Molecular Weight:	332
CAS Registry No.:	1861-32-1
PC Code:	078701

Physical Properties of DCPA

DCPA is a colorless or white crystal with a melting point of 155 °C and a bulk density of 0.75g/cm3. It has a relatively high octanol/water partition coefficient of 1.9×10^4 at 25 °C and a relatively low vapor pressure of 2.5×10^{-6} torr at 25° C. DCPA in the environment is not particularly persistent or mobile (Kd range from 5.56 to 70.3 ml/gram). DCPA is practically insoluble in water (0.5 ppm at 25 ^{o}C), but is readily soluble in most organic solvents.

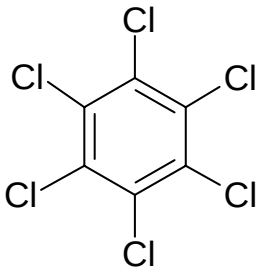
DCPA Metabolites of Toxicological Concern

DCPA is metabolized to mono-methyl-tetrachloroterephthalic acid (MTP) and tetra-chloroterephthalic acid (TPA) by hydrolysis of one or both of the methyl groups. This hydrolysis occurs both in animals and in soil. The metabolite TPA is most commonly found in the environment after DCPA use. The structures of these metabolites are shown below.

DCPA	MTP	TPA
		

DCPA Impurities of Toxicological Concern

Hexachlorobenzene (HCB) is an impurity of DCPA that is of toxicological concern because it is a suspect carcinogen. Product chemistry data (MRID 452467-01) indicate that the new DCPA technical has an HCB upper certified limit of 40 ppm. According to the 1998 RED, the maximum allowable HCB impurity level was 0.3 percent (3000 PPM). The structure of HCB is shown below:



3.0 HAZARD CHARACTERIZATION

3.1 Hazard Profile

Acute Effects

The acute toxicity of DCPA is low. It is a mild irritant to the eyes and skin and it is not a skin sensitizer. The acute toxicity values are included in Table 2.

Table 2 - Acute Toxicity of DCPA			
Guideline	**Study Type**	**Results**	**Tox Category**
81-1	Acute Oral - rat	LD50 >5000 mg/kg	IV
81-2	Acute Dermal - rabbit	LD50 >2000 mg/kg	III
81-3	Acute Inhalation - rat	LC50 >4.48 mg/L	III
81-4	Eye irritation - rabbit	mild irritation	III
81-5	Dermal irritation - rabbit	mild irritation	III
81-6	Dermal sensitization - guinea pig	not sensitizing	–

Non-Cancer Effects

The subchronic/chronic toxicity profile for DCPA is included in Appendix A. The most significant effects seen in the toxicity studies include decreased thyroid hormone production, thyroid histological changes and liver hypertrophy. The liver effects did not represent severe toxicity but are believed to be a precursor event for thyroid changes. The liver effects occur at much lower doses than the thyroid effects.

Liver enzymes were elevated in the chronic mouse and rat studies. In rats, hepatocyte centrilobular hypertrophy or swelling were noted in the subchronic, reproductive, and chronic feeding studies. In mice, centrilobular hepatocyte enlargement, classified "minimal", was seen only at the high dose in both the subchronic mouse study and in the chronic mouse study.

Thyroid toxicity in the chronic rat study consisted of decreased levels of thyroxine, follicular cell hyperplasia/hypertrophy, and increased thyroid weight. Thyroid follicular cell hyperplasia or hypertrophy occurred in the subchronic rat feeding studies and in the reproduction study. Thyroid hormones were not evaluated in the subchronic rat studies. Thyroid histological changes were not seen in the chronic mouse study.

Kidney weight was increased in the 90-day and chronic rat studies. There were increased incidences of chronic nephropathy in the chronic rat study and the reproduction study, along with increases in BUN and creatinine in the chronic rat study. There was anemia in the chronic rat study. An increased incidence of pneumonitis, diagnosed by histopathology, was seen in the chronic rat study and the reproduction study.

In the developmental toxicity study in rats, no developmental or maternal toxicity occurred at the high dose of 2000 mg/kg/day. In the developmental toxicity study in rabbits, no developmental toxicity occurred at a dose that caused maternal mortality (500 mg/kg/day). There are no concerns for either quantitative or qualitative susceptibility in the developmental studies.

In the 2-generation reproduction study, the parental NOAEL is 50 mg/kg/day and the parental LOAEL is 250 mg/kg/day based upon body weight decrements, gross and microscopic changes in kidneys and lungs, and microscopic changes in thyroids. There was no effect upon reproductive indices and the reproductive NOAEL is $\geq$1000 mg/kg/day, the highest dose tested. The offspring NOAEL is 50 mg/kg/day and the offspring LOAEL is 250 mg/kg/day based upon pup body weight decrements. The data from this study indicated no quantitative or qualitative susceptibility because the offspring NOAEL is the same as the parental NOAEL. The parental

effects, especially renal and thyroid toxicity, were more severe than the reversible effects upon pup body weight. In addition, the food consumption data show that pups were consuming approximately twice as much compound as were adults due to the greatly increased food consumption in the growing pups during this time.

There were no significant sex differences in metabolism of DCPA. Absorption was more efficient at 1 mg/kg/day (79%-86% of administered dose) than at 1000 mg/kg/day (6-9%). Compound was excreted in the urine as the mono-acid metabolite. Approximately 1% of compound in the urine was the di-acid metabolite and no parent compound was found in urine. Less than 1% was found in bile, so compound in feces represents unabsorbed compound. Although a high percentage of the administered dose was found in fat 12 hours after discontinuance of dosing (12% of dose in low-dose animals), radiolabel had rapidly depleted by 168 hours (0.03%). Concentration of radiolabel in the thyroid increased at 36 hours postdosing when compared to the 12 hour time period, however, radiolabel in the thyroid rapidly depleted by 168 hours. By 168 hours, highest concentration of radiolabel in both dose groups was in the kidney.

Cancer Effects

The following mutagenicity studies were negative: two Mouse lymphoma assays, a cytogenetic assay in CHO cells, two unscheduled DNA synthesis assays, and an assay for sister chromatid exchange in CHO cells. The Ames test study was classified as unacceptable.

Thyroid follicular cell adenomas were increased in male and female rats; thyroid follicular cell carcinomas were increased in female rats. Hepatocellular adenomas, hepatocellular carcinomas and hepatocholangiocarcinomas were increased in female rats. Hepatic adenomas were increased in female mice. Some phthalate compounds have been identified as peroxisome proliferators, although a study to evaluate this in DCPA has not been performed.

3. 2 Dose Response Assessment and Endpoint Selection

On April 11 and May 7, 2002, the Health Effects Division (HED) Hazard Identification Assessment Review Committee (HIARC) reviewed the recommendations of the toxicology reviewer for DCPA with regard to toxicological endpoints for use in occupational/residential exposure risk assessments. The HIARC concluded that the toxicology database for DCPA is not complete, however, it was adequate for selecting toxicity endpoints for risk assessment. The main uncertainty is extrapolation of the thyroid effects observed during the chronic study to short and intermediate exposure durations because thyroid hormones were not measured during the 28 day dermal and 90 day feeding studies. No doses were established for acute dietary exposures because no appropriate endpoint from a single exposure was identified in any of the toxicity studies. No doses were established for short/intermediate term dermal exposures because no systemic toxicity occurred in the dermal toxicity study at the high dose of 1000 mg/kg/day. The endpoints are included in Table 3 and were assessed using uncertainty factors of 10X for interspecies extrapolation and 10X and intraspecies variability.

The HIARC recommended that the toxicity endpoints for oral, dermal and inhalation exposures be aggregated as follows: chronic dietary, dermal, and inhalation exposure may be combined because the same study and endpoints were used for these routes. Short and intermediate term inhalation may be combined with short and intermediate term incidental oral exposure because the same study and endpoint was used for these routes and time periods.

The Carcinogenicity Peer Review Committee classified DCPA as Group C, possible human carcinogen. The **Q1* for DCPA**, based on the three combined liver tumors in female rats, and using a 3/4's scaling factor is 1.5×10^{-3}. HCB is a contaminant of DCPA with an upper tolerance limit of 0.3%. The HCB level in the study that was used to establish the Q1* for DCPA was 0.13 percent. HCB is classified as a B2, probable human carcinogen. The **Q_1^* for HCB** is 1.0 $(mg/kg/day)^{-1}$ based on hepatocellular carcinomas in female SD rats.

Because the cancer studies were performed via the dietary route of exposure, it is not known if the liver tumors would occur if exposures occurred via dermal exposure. In the absence of information to the contrary, it was assumed that the dermal exposure would contribute to the cancer risk in a linear fashion according to the absorbed dose. The dermal absorption rate had been quantified in a dermal absorption study and this rate was used to calculate a dermal dose. The dermal dose was added to the inhalation dose for handler exposures and was considered alone for post application exposures.

Table 3 - DCPA Toxicological Endpoints for Use in Human Health Risk Assessment		
Exposure Scenario	**Dose (mg/kg/day)**	**Endpoint for Risk Assessment**
Dietary Exposures (The uncertainty factor is 100 is for all scenarios. The FQPA factor is 1X)		
Acute	NOAEL = N/A Acute RfD = N/A	No appropriate endpoint from a single exposure was identified in any of the toxicity studies.
Chronic All populations	NOAEL = 1 Chronic RfD = 0.01 Chronic PAD = 0.01	Combined chronic/carcinogenicity study in rats. LOAEL = 10 mg/kg/day based on decreased thyroxine levels and liver and thyroid histological changes in males
Non-Dietary Exposures (An MOE of 100 is required for all scenarios. The FQPA factor is 1X)		
Incidental Oral Residential Only Short and Intermediate Term (1day - 6 months)	NOAEL = 50	90-day feeding study in rats. LOAEL = 100 mg/kg/day based on increased incidence of hepatocellular hypertrophy
Dermal Short and Intermediate Term	NOAEL = N/A	Quantitation not required. No systemic toxicity occurred in the dermal toxicity study at the high dose of 1000 mg/kg/day. Although the thyroid was not evaluated in this study, no liver effects were noted and liver hypertrophy is believed to be a precursor event for thyroid changes.
Dermal Long Term	Oral NOAEL = 1	Combined chronic/carcinogenicity study in rats. LOAEL = 10 mg/kg/day based on decreased thyroxine levels and liver and thyroid histological changes in males
Inhalation Short and Intermediate Term (1day to 6 months)	Oral NOAEL = 50	90-day feeding study in rats. LOAEL = 100 mg/kg/day based on increased incidence of hepatocellular hypertrophy
Inhalation Long-Term (>6 Months)	Oral NOAEL = 1	Combined chronic/carcinogenicity study in rats. LOAEL = 10 mg/kg/day based on decreased thyroxine levels and liver and thyroid histological changes in males.
DCPA Cancer	Classification: Group C, possible human carcinogen. Q1* = 0.0015 $(mg/kg/day)^{-1}$ based upon three combined types of liver tumors in female rats.	
HCB Cancer	Classification: Group B, probable human carcinogen. Q1* = 1.0 $(mg/kg/day)^{-1}$ based upon liver tumors in female rats.	
DCPA Dermal Absorption	22%	Ten hour value after exposure to 47.5 ug/cm^2.
HCB Dermal Absorption	27%	Ten hour value after exposure to 0.95 ug/cm^2.

3.3 Data Gaps and Requirements

A guideline 28-day inhalation study in rats, which must include assessment of thyroid histopathology and thyroid hormone levels, is required.

A confirmatory study showing the comparative short-term thyroid toxicity of DCPA in adults and offspring is required. This study should include evaluation of thyroid hormone levels and liver induction. The registrant should consult with the Agency to discuss this study before beginning the study.

3.4 FQPA Considerations

The FQPA Safety Factor Committee evaluated the available hazard and exposure data for DCPA on May 13, 2002 and recommended that the FQPA safety factor to be used in human health risk assessments (as required by Food Quality Protection Act of August 3, 1996) be reduced to1x for the following reason:

- The toxicology database for DCPA contains acceptable rat and rabbit developmental studies and an acceptable 2-generation rat reproduction study; a developmental neurotoxicity study is not required. The HIARC concluded that there is not a concern for pre- and/or postnatal toxicity resulting from exposure to DCPA. Although comparative thyroid measurements have been requested, a database uncertainty factor is not necessary since this information is considered to be confirmatory. There are no residual concerns since the endpoints selected for dietary and non-dietary exposure assessment are protective of the observed thyroid toxicity.

- There are no residual uncertainties identified in the exposure databases. The dietary food exposure assessment is based on average field trial values corrected by percent crop treated. Modeling results are used to calculate Estimated Environmental Concentrations (EECs) for drinking water risk assessment. Submitted turf transferable residue (TTR) data will be used along with the Residential SOPs to assess post-application exposure to children as well as incidental oral exposure of toddlers. These assessments will not underestimate the exposure and risks posed by DCPA.

A summary of the FQPA factors for DCPA is included in Table 4.

Table 4 - Summary of FQPA Safety Factors for DCPA				
	LOAEL to NOAEL (UF_L)	**Subchronic to Chronic (UF_S)**	**Incomplete Database (UF_{DB})**	**Special FQPA Safety Factor (Hazard and Exposure)**
Magnitude of Factor	1X	1X	1X	1X
Rationale for the Factor	No LOAEL to NOAEL extrapolations performed	No subchronic to Chronic extrapolations performed	Database is sufficiently complete to assess risks to infants and children.	No residual concerns regarding pre- or post-natal toxicity or completeness of the toxicity or exposure databases
Endpoints to which the Factor is Applied	Not Applicable	Not Applicable	Not Applicable	Not Applicable

3.5 Endocrine Disruption

EPA is required under the FFDCA, as amended by FQPA, to develop a screening program to determine whether certain substances (including all pesticide active and other ingredients) "may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or other such endocrine effects as the Administrator may designate." Following the recommendations of its Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC), EPA determined that there was scientific bases for including, as part of the program, the androgen and thyroid hormone systems, in addition to the estrogen hormone system. EPA also adopted EDSTAC's recommendation that the Program include evaluations of potential effects in wildlife. For pesticide chemicals, EPA will use FIFRA and, to the extent that effects in wildlife may help determine whether a substance may have an effect in humans, FFDCA authority to require the wildlife evaluations. As the science develops and resources allow, screening of additional hormone systems may be added to the Endocrine Disruptor Screening Program (EDSP).

When the appropriate screening and/or testing protocols being considered under the Agency's EDSP have been developed, DCPA may be subjected to additional screening and/or testing to better characterize effects related to endocrine disruption.

DCPA treatment caused decreased levels of thyroid hormones and compensatory thyroid hyperplasia and/or hypertrophy in rats. Thyroid effects were accompanied by hepatocyte hypertrophy and/or hepatocyte swelling. The liver effects did not represent severe toxicity but are believed to be a precursor event for the thyroid changes. This mechanism is stimulation of liver microsomal enzymes resulting in increased metabolism of thyroid hormones. This results in thyroid hyperplasia as the thyroid produces more hormones in compensation for the decreased hormone levels.
DCPA shares some structural similarities with phthalate plasticizers which cause anti-androgenic

effects in lab animals. However, DCPA is a terephthalate, and does not share the key structure necessary for anti-androgenic properties. The structure common to phthalates with anti-androgenic properties is a benzene ring with paired ester groups, C4-C6 in length, in ortho position. Phthalates with ester groups in para positions, or with ester groups of C2 or shorter in length, did not have anti-androgenic properties (LEG Gray, *et al*. Toxicol. Sci. 58:350-365. 2000). DCPA has two esters of C1 length in para position, and lacks the structure in common with the anti-androgenic phthalates.

4.0 NON-OCCUPATIONAL EXPOSURE ASSESSMENT

4.1 Summary of Registered Uses

DCPA is currently marketed as only one product, Dacthal W-75 (EPA Reg. #5481-490) which is produced by the AMVAC Chemical Corporation. Dacthal W-75 is a wettable powder formulation which contains 75% DCPA as the active ingredient. It also contains trace amounts (less than 40 ppm) of hexachlorobenzene (HCB) as a manufacturing impurity. Dacthal was previously manufactured by ISK Biosciences until it was discontinued in 1997. AMVAC began producing Dacthal W-75 in 2001. The other DCPA products as listed in Table 5 are currently in the process of product re-registration. According to the DCPA RED, which was published in 1998, all of the uses except turf are eligible for registration.

Table 5 - DCPA Products and Application Rates (lb ai/acre)

Product	Formulation	Reg Number	Turf Rate	Ornamental Rate	Vegetable Rate
Dacthal W-75	Wettable Powder	5481-490	10/15	10.5 to 12	4.5 to 10.5
Lebanon Pre-Emergence Weed Control	Granular	961-273	10/15	8.7	8.2 to 10.3
ACME Garden Weed Preventer Granules	Granular	33955-474	N/A	10.9	9.1
Dacthal G-5	Granular	5481-489	10/15	9.0	8.0 to 10.5
Dacthal Flowable Herbicide Turf Care	Liquid	5481-487	10/15	12	N/A
PBI Garden Weeder	Granular	2217-617	N/A	10.9	9.1

DCPA is used as a selective herbicide for pre-emergence application to kill crabgrass and certain broad-leafed weeds on mineral soils in brassica, bulb vegetables, cucurbits, potatoes, fruiting vegetables, strawberries, ornamentals and turf. DCPA is proposed to be applied to the soil prior to crop seed germination when used to control weeds on fields planted with minor crops in CA (basil, celeriac, chicory, chives, coriander, dill, marjoram, oriental radish, radicchio and parsley) and in WI and NC (ginseng). The proposed application rate for all crops is 4.5 to 10.5 lb ai per acre. When used for other crops, DCPA can be applied at seeding or at transplant for early season weed control and can be applied over the top for later season control. The application rates for the other crops also range from 4.5 to 10.5 lbs ai/acre. Typically only one application is made, but a second application can be made at layby to bulb vegetables and potatoes. When used on turf to control crabgrass, DCPA is applied at a rate of 10.5 lbs ai/acre in the early

spring before the weeds germinate. A second application at 5.25 lbs ai/acre can be made two months later if necessary. When used to control annual blue grass, DCPA is applied at 15 lbs ai/acre in the late summer or early fall before weed germination. Both the crab grass and the blue grass treatments can be made in the same season.

4.2 Dietary Exposure/Risk Pathway

Potential dietary exposure to DCPA in the diet occurs through food and water. Data supporting food exposure are adequate and are summarized in the anticipated residue memorandum which was prepared by W. Hazel (6/13/02; DP 283274).

4.2.1 Dietary Exposure - Food

Tolerances for residues of DCPA in/on plant commodities [40 CFR §180.185] are expressed in terms of the combined residues of DCPA and its monodemethylated (MTP) and didemethylated (TPA) metabolites. Plant commodity tolerances range from 0.05 ppm in corn grain to 15 ppm in radish tops. Tolerances in livestock commodities have not yet been established although a ruminant metabolism study and a poultry feeding study indicate that tolerances are necessary. The dietary exposure assessments include expected residues of DCPA in livestock commodities. An adequate method is available for the enforcement of tolerances as currently defined.

The qualitative nature of the residue in plants is adequately understood based on acceptable studies on onions, turnips, and tobacco. The metabolism of DCPA in plants is via ester hydrolysis. The residues of concern in plants are DCPA and its metabolites MTP and TPA which are currently regulated and which are also the residues of toxicological concern. Studies conducted with onion and turnip indicate that the impurity HCB is not metabolized appreciably in these plants.

The nature of the residue in ruminants is adequately understood. DCPA, MTP, and TPA are the residues of concern. Until adequate cattle feeding studies are available, the data from the ruminant metabolism study will be used to estimate residues in meat and milk commodities. The requirement for a poultry metabolism study has not been met, and remains in effect. Until these data are generated, EPA will use the existing poultry feeding studies for exposure/risk assessment based on the assumption that the residues of concern in poultry tissues and eggs are the same as those delineated in meat and milk from the acceptable ruminant metabolism study. Based on these metabolism studies, the MARC has determined that the residue of concern in plant and livestock commodities for purposes of both regulation (tolerance expression) and risk assessment continues to be combined residues of DCPA, MTP, and TPA calculated as DCPA (W. Hazel. 5/15/02. D282838).

Adequate methodology is available to enforce existing and proposed tolerances in/on plant commodities. Three tolerance enforcement methods for plant commodities are published in FDA's Pesticide Analytical Manual (PAM), Vol. II (Section 180.185), as Methods A, B, and C. Residue data submitted in response to the 6/88 Guidance Document were collected using GC/EC methods similar to the PAM, Vol. II methods. These methods are adequate for collection of DCPA, HCB, MTP, and TPA residue data from crops (including processed commodities). The limits of detection (LOD) are 0.01 ppm each for DCPA, MTP, and TPA, and 0.0005 ppm for HCB. These methods are suitable candidates for validation procedures as enforcement methods for plant commodities.

Another GC/EC method, similar to those submitted for plants, is available for determining DCPA, MTP, and TPA in milk and beef fat. Recoveries of each compound using 12 samples each of milk and beef fat fortified at 0.01-5 ppm were acceptable. The LOD is 0.01 ppm. This method is suitable for Agency validation and inclusion in PAM, Vol. II pending successful independent laboratory validation. The registrant has indicated that independent laboratory validation of the method is underway. The registrant must submit independent laboratory validation data for enforcement method(s) for animal commodities and submit the method(s) for Agency validation and inclusion in PAM, Vol. II. Representative samples from adequate animal metabolism studies must be analyzed by preferred enforcement method(s) to ascertain their ability to adequately recover and quantify DCPA, MTP, and TPA.

DCPA *per se* is completely recovered using PAM, Vol. I Multiresidue Protocols D and E (PESTDATA, PAM, Vol. I, Appendix, 8/93). Data submitted by the registrant indicate that TPA is not recovered by Protocols B and C. Multiresidue testing data on MTP are not available.

The final report for a 4-year storage stability study on DCPA support the conclusion that residues of DCPA, MTP, TPA and HCB are stable in frozen samples of broccoli, onion bulbs, celery, snap beans, bell peppers, and sweet potatoes stored for 4 years. However, the registrant must submit storage intervals and conditions for field trial samples analyzed in MRIDs 00017975, 00018299, 00033087, 00038919, 00058377, 00058378, 00072099, 00090259, 00114643, 00114678, 00114679, 00114680, 00114681, 00121864, and 00130562. If it can be confirmed that samples from these earlier field trials had been stored frozen and for durations not significantly longer than 4 years, all field trial data will be considered to be fully validated by the 4-year storage stability study.

The available field trial data for all commodities having DCPA tolerances have been reevaluated for purposes of tolerance reassessment. Overall, acceptable field trials reflecting the maximum registered use patterns and conditions under which the pesticide could be applied were conducted. The geographic representation for each commodity is generally adequate, and a sufficient number of trials reflecting the representative WP formulation class was conducted.

The following additional data were required in the 1998 RED for confirmatory purposes: ruminant feeding study; poultry metabolism study; additional ILV testing and radiovalidation of the livestock method; storage time and temperature of samples from several older field trial studies; and large-scale field rotational crop studies on representative crops to which the

registrant wishes to permit rotation. These data are not required for this assessment of new uses, however, because these uses do not include animal feed crops.

There are no Codex MRLs for DCPA residues; therefore, compatibility issues do not exist. However, there are Canadian MRLs ranging from 1-5 ppm in/on leafy crops, cole crops, cucurbits, legumes, root crops, fruiting vegetables, bulb vegetables, and strawberries; the Canadian MRLs appear to include only the parent compound but, numerically, they are identical to U.S. tolerances.

The U.S. FDA monitoring data and USDA PDP survey data are of limited usefulness because samples were not analyzed for the metabolites MTP and TPA which comprise a significant, yet variable, proportion of the total residue of concern. Field trial data and tolerances were used to arrive at the values for DCPA residues in virtually all cases (refer to anticipated residue memorandum for details). Quantitative usage (percent crop treated) information is taken from a usage analysis conducted by F. Hernandez of OPP/BEAD dated 5/22/02.

The Agency has high confidence in the nature of the residue in plants and the quality of the field trial residue data. We are confident that these data do not underestimate dietary exposure to the residues of concern of DCPA. Although livestock commodities are not expected to contribute significantly to dietary risk, a ruminant feeding study and a hen metabolism study are necessary to confirm the poultry residues of concern, to establish meat, milk, poultry, and egg tolerances, and to refine dietary exposure. Although a Tier 2/3 dietary risk assessment was conducted and is the most refined assessment to date for DCPA, there are some uncertainties associated with the exposure estimates as follows: (i) use of field trial data for most commodities will overestimate exposure; (ii) no cooking studies were used; (iii) use of tolerance level residues and 100% crop treated for several minor crops and all proposed crop sites; (iv) DEEM default processing factors were used in the assessment except for tomatoes; (v) use of the ruminant metabolism study to calculate anticipated residues; (vi) in the absence of a hen metabolism study, the assumption that poultry, ruminants, and rats metabolize DCPA similarly; and (vii) uncertainty in extrapolating exposures for certain population subgroups which may not be sufficiently represented in the DEEM consumption surveys.

4.2.2 Acute Dietary

No adverse effects reflecting a single dose were identified; therefore, no acute endpoint was selected. An acute dietary risk assessment was not conducted.

4.2.3 Chronic Dietary

DCPA chronic dietary exposure assessments were conducted using the Dietary Exposure Evaluation Model (DEEM™) software Version 7.76, which incorporates consumption data from USDA's Continuing Surveys of Food Intake by Individuals (CSFII), 1989-1992. The 1989-92 data are based on the reported consumption of more than 10,000 individuals over three consecutive days, and therefore represent more than 30,000 unique "person days" of data. Foods "as consumed" (e.g., apple pie) are linked to raw agricultural commodities and their food forms (e.g., apples-cooked/canned or wheat-flour) by recipe translation files internal to the DEEM software. Consumption data are averaged for the entire U.S. population and within population subgroups for chronic exposure assessment.

For chronic exposure and risk assessment, an estimate of the residue level in each food or food-form (e.g., orange or orange-juice) on the commodity residue list is multiplied by the average daily consumption estimate for that food/food form. The resulting residue consumption estimate for each food/food form is summed with the residue consumption estimates for all other food/food forms on the commodity residue list to arrive at the total estimated exposure. Exposure estimates are expressed in mg/kg body weight/day and as a percent of the chronic population adjusted dose (cPAD). The cPAD is the Rfd divided by the FQPA safety factor (which is 1 for DCPA). This procedure is performed for each population subgroup.

Chronic dietary exposures were calculated using anticipated residues for currently labeled crops, tolerance levels for the new uses and percent crop treated data provided by the Biological and Economics Analysis Branch. The percent crop treated data was used for currently labeled crops while an assumption of 100 percent crop treated was used for the new uses. These exposures were compared to the cPAD of 0.01 mg/kg/day and the risks were significantly below HED's level of concern ($\leq$1.1% cPAD) for all population subgroups as shown in Table 6.

Table 6. Chronic Dietary Exposure Summary for DCPA

Population	Exposure (mg/kg body wt/day)	Percent cPAD
U.S. Population	0.000097	1.0
All Infants	0.000085	0.9
Children (1-6 yrs old)	0.000113	1.1
Children (7-12 yrs old)	0.000130	1.1
Females (13-50 yrs old)	0.000088	0.9
Males (13-19 yrs old)	0.000064	0.6
Males (20+ yrs)	0.000094	1.0
Seniors (55+ yrs)	0.000108	1.1

4.2.4 Cancer Dietary

The lifetime average daily dose of DCPA in the diet of the general population is estimated to be 0.000097 mg/kg/day using the same inputs as for the chronic dietary exposures. This dose multiplied times the Q_1* of 1.5 x 10^{-3} yields a lifetime cancer risk of 1.5 x 10^{-7}. This risk is not of concern.

4.3 Water Exposure/Risk Pathway

The Environmental Fate and Effects Division provided the drinking water assessment using simulation models to determine the estimated environmental concentration (EEC) of DCPA in ground and surface water. Biodegradation is the primary dissipation process for DCPA with approximately 20 to 40% of the loss also occurring by volatilization. Parent DCPA is not especially persistent or mobile (K_d range from 5.56-70.3 mL/g). Substantial amounts of DCPA could be available for runoff for several weeks post-application. Most DCPA runoff will generally occur in the form of adsorption to eroding soil as opposed to dissolution in runoff water. DCPA could be somewhat persistent in many surface waters, particularly those with low microbiological activities and long hydrological residence times.

Tetrachloroterephthalic acid (TPA or di-acid) is the only significant DCPA metabolite, with monomethyl tetrachloroterephthalic acid (mono-acid) as a minor metabolite. TPA is unusually mobile (K_d 0.08-0.19 mL/g) and persistent (no loss of TPA in an aerobic soil metabolism study after 300 days). Data suggest that TPA will leach to groundwater wherever DCPA is used, regardless of soil properties. TPA appears to be substantially more persistent than parent DCPA and exhibits low soil/water partitioning. Therefore, substantial quantities of TPA should be available for runoff for a longer period than the parent DCPA.

Surface Water Modeling PRZM 3.12/ EXAMS 2.7.97 modeling was performed with index reservoir (IR) scenarios and percent cropped area (PCA) adjustment factors. Two different crop scenarios; cotton in Mississippi and Turf in Pennsylvania were chosen to estimate the concentration of DCPA and the metabolite TPA in surface drinking water. These scenarios were chosen to represent a geographically dispersed range of modeled surface water concentrations in areas representative of where DCPA is heavily used (Northeast states on turf) or has the potential for heavy runoff (Southern states on cotton). Default percent crop area (PCA) adjustment factors were applied. The results of this modeling is included in Table 7.

Table 7. Estimated Environmental Concentrations of DCPA and TPA in the Index Reservoir Using PRZM-EXAMS						
Crop and Location	**Application Rate**	**Scenario**	**Annual Mean DCPA EEC[A]**	**Annual Mean TPA EEC[A]**	**36 Year Mean DCPA EEC[B]**	**36 Year Mean TPA EEC[B]**
Turf, PA	15 lb ai/acre	Index Reservoir	48.2 ug/liter	369 ug/liter	38 ug/liter	184 ug/liter
		Index Reservoir (PCA = 0.87)	42*	321*	33.1*	160*
Cotton, MS	10.5	Index Reservoir	56	1829	40	1101
		Index Reservoir (PCA = 0.2)	11.2*	366*	8.0*	220*
A. Maximum estimated environmental concentration (EEC) predicted to occur in a ten year period. B. Average of annual means over a thirty six year period.						

* Used to calculate DWLOC values.

Surface Water Monitoring data. The are no surface water monitoring data available for TPA. There are limited surface water monitoring data available for DCPA from the US Geological Survey (USGS) National Water Quality Assessment Program (NAWQA). NAWQA collected 1557 surface water samples from 62 agricultural stream sites, 609 samples from 22 urban stream sites and 592 samples from 31 integrator sites (on large rivers and streams) during the period from 1992-1998. One to two samples was collected each month during periods when pesticide transport in the streams was expected to be low and 1 to 3 samples was collected per week during periods when elevated levels of pesticides were expected. The results of this monitoring are included in Table 8 .

Table 8 - USGS NAWQA Surface Water Data for DCPA 1992 to 1998						
Site Type	Number of Sites	Number of Samples	Frequency of Detection[1]	50th percentile (ug/liter)	95th percentile (ug/liter)	Maximum (ug/liter)
Agricultural Streams	62	1557	18	<0.002	0.02	40
Urban Streams	22	609	27	<0.002	0.01	0.05
Large Rivers and Streams (Integrator Sites)	31	592	24	<0.002	0.01	0.18
1. The limit of detection is 0.002 ug/liter.						

According to EFED, the DCPA surface water data are not adequate to perform a quantitative drinking water assessment for regulatory purposes because the frequency of sampling and the length of sampling period were not sufficient to represent the temporal and spatial variation in residue levels.

Ground Water Modeling: SCI-GROW modeling was used to estimate the concentration of DCPA and TPA in drinking water from shallow ground water sources. The model estimates upper-bound ground water concentrations of pesticides likely to occur when the pesticide is used at the maximum allowable rate in areas where ground water is vulnerable to contamination. Although SCI-GROW, unlike the PRZM/EXAMS surface water models, does not require a specific crop scenario, the same application rates were used as for the surface water modeling. The SCI-GROW model estimated the concentration of DCPA in drinking water from shallow ground water sources to be **0.014 µg/liter for turf** and **0.0099 ug/liter for cotton**. The SCI-GROW model estimated the concentration of TPA in drinking water to be **275 µg/liter for turf** and **192 ug/liter for cotton.** These estimated concentrations represent 90 day average values.

Groundwater Monitoring Data

EFED has limited targeted monitoring data on the concentrations of DCPA and its degradates in groundwater. These data are summarized in Table 9.

Detections of parent DCPA have been reported. This is contrary to the environmental chemistry and environmental fate data of parent DCPA which indicate that parent DCPA would not be mobile. TPA is the major degradate found in ground water. DCPA or TPA and the mono-acid degradate have been detected in groundwater in 24 states. Concentrations of DCPA degradates ranged from trace levels to 1477 ppb. The maximum reported DCPA concentration is 7.7 ppb.

According to EFED, the ground water data are not adequate to perform a quantitative drinking water assessment for regulatory purposes because the frequency of sampling and the length of sampling period were not sufficient to represent the temporal and spatial variation in residue levels.

Table 9 - Groundwater Monitoring Data for DCPA and TPA

Analyte	Concentration (ppb)	Area Sampled	Data Source
DCPA	Not detected	N=1347	National Pesticide Survey, EPA 1990
TPA	0.34 ppb (med) to 7.2 ppb (max)	CWS[1] (n=564)	
TPA	0.38 ppb (med) to 2.4 ppb(max)	RDW[2] (n=783)	
DCPA	Detected in 157 wells.	3570 Wells	State Ground Water Monitoring Studies
TPA	Detected in 151 wells. Max values were generally <15 ppb, but higher levels were found in NY (1039 ppb) and OR (986 ppb). Mean and median values in NY were 109 and 13.2 ppb.	1341 Wells	
DCPA	Detected in 5 samples	2033 wells	Pesticides in Ground water Database, EPA 1992
DCPA	0.1 to 0.35 ppb (NY) 0.1 to 0.2 ppb (CA)	Wells near onion fields in CA and turf fields in NY	MRID 436464-01 - Ground Water Monitoring Study

Table 9 - Groundwater Monitoring Data for DCPA and TPA			
Analyte	**Concentration (ppb)**	**Area Sampled**	**Data Source**
TPA	Detected in 12% of wells in NY and 29% of wells in CA		
DCPA	Detected in 0.32% of samples. (Max concentration = 0.002 ppb)	1849 wells in major aquifers	National Water Quality Assessment, USGS 1998
1. CWS = Community Water System 2. RDW = Rural Drinking Water Well			

4.4 Residential Exposure/Risk Pathway

4.4.1 Residential Handlers

The inhalation exposures of residential handlers mixing/loading and applying DCPA to lawns and gardens was assessed using maximum label rates and standard SOP assumptions. These exposures were compared to the short/intermediate term oral endpoint of 50 mg/kg/day as listed in Table 3 and inhalation absorption was assumed to be equivalent to oral absorption. Dermal exposures were not assessed for non-cancer effects because a dermal endpoint for short term exposures was not selected. Chronic exposures were not assessed because DCPA is only applied a few times per year. The non-cancer MOEs were all above the target MOE of 100 as shown in Table 10.

Table 10 - DCPA Non-Cancer Risk for Homeowner Applications				
Exposure Scenario	**Crops**	**Application Rate (lb ai/Acre)**	**Treated Area (Acre/day)**	**DCPA MOE**
1 - Apply Granules by Hand or Shaker Can	Garden Vegetables	10.5	0.023	31000
2 - Load/Apply Granules with a Belly Grinder	Garden Vegetables	10.5	0.023	230000
3A - Load/Apply Granules with a Broadcast Spreader	Garden Vegetables	10.5	0.500	7.3 Million
3B - Load/Apply Granules with a Broadcast Spreader	Lawns	15.0	0.500	5.1 million
Note - 1000 square feet equals 0.023 acre.				

The cancer risks for the residential handler scenarios were calculated assuming two applications per year and are shown in Table 11. Both the dermal and inhalation routes of exposure were added to determine the lifetime average daily dose. None of the scenarios are of concern for either DCPA or HCB because the cancer risks are below 1.0×10^{-6}.

Table 11 - Residential Handler Cancer Risks of DCPA and HCB (Assuming Two Application per Year)					
Exposure Scenario	Crop	Application Rate (lb ai/acre)	Area Treated (Acre/Day)	DCPA Cancer Risk	HCB Cancer Risk
1 - Apply Granules by Hand or Shaker Can	Garden Vegetables	10.5	0.023	5.2e-07	1.7e-08
2A- Load/Apply Granules with a Belly Grinder	Garden Vegetables	10.5	0.023	4.9e-07	1.6e-08
3A - Load/Apply Granules with a Broadcast Spreader	Garden Vegetables	10.5	0.50	6.6e-08	2.2e-09
3B - Load/Apply Granules with a Broadcast Spreader	Lawns	12.5	0.50	7.8e-08	2.6e-09
Note - 1000 square feet equals 0.023 acre.					

4.4.2 Residential Post Application Exposure and Risk

Garden Vegetables

Significant post application exposures are not anticipated for garden vegetables because the applications are made to freshly cultivated soil using only the granular products. The risks of acute oral exposures due to granular ingestion by children were not assessed because no endpoint for acute dietary exposures was selected by the HIARC (no adverse effects were seen following a single dose).

Turf

Significant post application exposures are anticipated for turf because broadcast applications are made to prevent the growth of weeds throughout the lawn. These exposures are anticipated to be short term because only one or two applications are made per growing season and the label recommended application interval is two months or longer. Only incidental oral exposures were assessed for toddlers because a dermal endpoint for short/intermediate term exposures was not selected. Cancer risks were assessed only for adults per Agency policy and were assessed only for the dermal exposure pathway.

A Turf Transferable Residue study had been submitted in support of the RED. This study involved the application of Dacthal W-75 to Kentucky Bluegrass turf plots in Ohio. Three of the treated plots were irrigated with 0.5" water immediately following sampling at one hour after treatment and 0.18" of rain occurred at day after treatment (DAT) six. This study generally complied with series 875 guidelines and the DCPA data is of sufficient quality to be used for exposure and risk assessment purposes. This data is presented in Table 12 and indicated that irrigation reduced the residue from an initial value of 4.2 ug/cm^2 at DAT 0.04 to 1.6 ug/cm^2 at DAT 0.08. The residue then dissipated at rate of 6.1 percent per day from DAT 1 until the last day of the study (DAT 14).

Table 12 - Dissipation of DCPA Applied to Turf					
Analyte (Site)	**TTR (ug/cm^2) at DAT 0 Before Irrigation**	**TTR at DAT 0 After Irrigation (Note 1)**	**TTR at DAT 1**	**Correlation Coefficient (Note 2)**	**Half Life (days)**
DCPA (Irrigated) DCPA (Non-Irrigated)	4.2 ± 1.0 4.2 ± 1.5	1.6 ± 0.41 N/A	1.3 ±0.13 5.6± 4.3	0.90 (n=7) 0.19 (n=7)	11.0 N/A
Note 1 - Irrigation occurred after the day after treatment (DAT 0.04) sample and before the DAT 0.08 sample. The DAT 0.08 and DAT 0.25 samples were averaged to determine the DAT 0 TTR for the irrigated site. Note 2 - Regression calculations were performed using DAT 0.08 to DAT 14 data for the irrigated site and DAT 0.0 to DAT 7 for the non-irrigated site. These calculations indicated that statistically significant dissipation only occurred at the irrigated site.					

Standard SOP assumptions were used instead of the TTR data to calculate incidental oral exposure for toddlers exposed to turf. The TTR data were not used because they are not compatible with the incidental oral exposure pathways. The incidental oral exposures were compared to the NOAEL of 50mg/kg/day and a summary of MOEs is given in Table 13. All of the MOEs exceeded the target of 100 on the day after treatment zero (DAT 0) and are not of concern.

Table 13 - Incidental Oral MOEs for Toddler Post Application Turf Exposure					
DAT	**Application Rate**	**Hand to Mouth MOE**	**Object to Mouth MOE**	**Soil Ingestion MOE**	**Aggregate MOE**
0	15 lb ai acre	220	890	66000	180

The cancer risks for adults exposed to treated and irrigated turf were calculated using standard assumptions and the TTR data averaged over 14 days. The data were normalized to an average application rate of 12.5 lbs ai/acre. It was assumed four days of exposure to turf that was treated within 14 days would occur per year. These risks are shown in Table 14.

Table 14 - Cancer Risks for Adult Post Application Turf Exposure[1]					
Turf Transferable Residue Level[2](ug/cm^2)	**Days Per Year Exposure**	**DCPA LADD[3] (mg/kg/day)**	**DCPA Cancer Risk[4]**	**HCB LADD[3] (mg/kg/day)**	**HCB Cancer Risk[5]**
0.64 (DCPA) 0.0026 (HCB)	4	2.3e-04	3.4e-07	1.1e-08	1.1e-08
1. Average over 14 days after an application of 12.5 lb ai/acre immediately followed by irrigation. 2. Assuming heavy yardwork with a transfer coefficient (TC) of 7300 cm^2/hour. 3. LADD = TTR x TC x 0.001 mg/µg x DA x 2 hours exposure/day x (1/70 kg) x 4/365 x 50 years /70 years 4. DCPA Cancer Risk = LADD x Q_1* where Q_1* = 0.0015 mg/kg/day^{-1} for DCPA 5. HCB Cancer Risk = LADD x Q_1* where Q_1* = 1.0 mg/kg/day^{-1} for HCB					

4.4.3 Other Residential Exposures

This assessment for DCPA reflects the Agency's current approaches for completing residential exposure assessments based on the guidance provided in the *Draft: Series 875-Occupational and Residential Exposure Test Guidelines, Group B-Postapplication Exposure Monitoring Test Guidelines,* the *Draft: Standard Operating Procedures (SOPs) for Residential Exposure Assessment,* and the *Overview of Issues Related to the Standard Operating Procedures for Residential Exposure Assessment* presented at the September 1999 meeting of the FIFRA Scientific Advisory Panel (SAP). The Agency is, however, currently in the process of revising its guidance for completing these types of assessments. Modifications to this assessment shall be incorporated as updated guidance becomes available. This will include expanding the scope of the residential exposure assessments by developing guidance for characterizing exposures from other sources not addressed in this document such as from spray drift and exposures to farmworker children.

4.4.4 Spray drift

Spray drift is always a potential source of exposure to residents nearby to spraying operations. This is particularly the case with aerial application, but, to a lesser extent, could also be a potential source of exposure from ground application methods. The Agency has been working with the Spray Drift Task Force, EPA Regional Offices and State Lead Agencies for pesticide regulation and other parties to develop the best spray drift management practices. The Agency is now requiring interim mitigation measures for aerial applications that must be placed on product labels/labeling. The Agency has completed its evaluation of the new data base submitted by the Spray Drift Task Force, a membership of U.S. pesticide registrants, and is developing a policy on how to appropriately apply the data and the AgDRIFT computer model to its risk assessments for pesticides applied by air, orchard air-blast and ground hydraulic methods. After the policy is in place, the Agency may impose further refinements in spray drift management practices to reduce off-target drift and risks associated with aerial as well as other application types where appropriate.

5.0 AGGREGATE RISK ASSESSMENTS AND RISK CHARACTERIZATION

In examining aggregate exposure, FQPA directs EPA to take into account available information concerning exposures from pesticide residues in food and other exposures for which there is reliable information. These other exposures include drinking water and non-occupational exposures, e.g., to pesticides used in and around the home. Risk assessments for aggregate exposure consider short-, intermediate- and long-term (chronic) exposure scenarios considering the toxic effects which would likely be seen for each exposure duration. DCPA is a food use chemical. Drinking Water Levels of Comparison (DWLOC) have been calculated for DCPA. There are residential (non-occupational) uses of DCPA; therefore, the considerations for aggregate exposure are those from food, drinking water and residential exposure. The following assumption and factors were used in calculating aggregate risks and DWLOCs.

- Body weights (kg) of 70, 60, 10 and 10 were used for adults, adult females, children and infants.
- Water consumption values (liters per day) of 2 , 2, 1 and 1 were used for adults, adult females, children and infants.
- The short and intermediate term exposures were compared to the Oral NOAEL of 50 mg/kg/day divided by the uncertainty factor of 100 to determine the DWLOC.
- The estimated environmental concentrations (EEC) for DCPA and the metabolite TPA were added to yield the combined EEC. The MARC concluded that the metabolites such as TPA should be a part of the regulated residues and are assumed to be of equivalent toxicity.
- The DWLOCs for Short/Intermediate term and chronic exposures were compared to the annual mean combined EEC in surface water.
- The DWLOCs for cancer risks were compared to the 36 year mean of annual means combined EEC in surface water.

5.1 Acute Aggregate Risk

An acute endpoint was not identified by the HIARC; therefore, no acute aggregate risk assessment is required.

5.2 Short Term Aggregate Risk

Residential scenarios were identified for adults (Inhalation Exposure from Hand/Shaker Application of Granules to Home Gardens) and children (Incidental Oral Exposure on Treated Turf). These exposures are anticipated to be short term in nature because DCPA is applied only once or twice a year with an application interval of two months or longer. Short term DWLOCs for DCPA were calculated based on chronic food exposure plus the residential handler inhalation exposure for adults and the chronic food exposure plus the incidental oral exposure for toddlers. These are presented in Table 15 and indicate that the DWLOCs are substantially greater than the combined estimated environmental concentration (EEC). This means that the risk cup has sufficient room to accommodate the estimated water risk and the resulting short term aggregate risk is not of concern.

Table 15 - DCPA Summary of Short Term DWLOC Calculations

Population Subgroup	NOAEL (mg/kg/day) Divided by UF of 100	Food Exposure (mg/kg/day)	Incidental Oral Exposure for Toddlers on Treated Turf (mg/kg/day)	Inhalation Exposure for Adult Handlers (mg/kg/day)	Available Water Exposure[A] (mg/kg/day)	DWLOC[B] (ug/liter)	Surface Water Combined EEC[CD] (Annual Mean) (ug/liter)
U.S. Population	0.50	0.000097	N/A	0.0016	0.498	17441	363
Females 13-50 yrs	0.50	0.000088	N/A	0.0016	0.498	14949	363
Children 1-6 yr	0.50	0.000113	0.42	N/A	0.080	799	363
All Infants	0.50	0.000085	0.42	N/A	0.080	799	363

A. Available water exposure =(NOAEL/SF) - [Food Exposure - Incidental Oral Exposure (toddlers) or Handler Inhalation Exposure (adults)]
B. DWLOC (ug/liter) = [Available water exposure x body weight x 1000 ug/mg]/[water consumption (liter)]
C. Surface Water Combined EEC = Annual Mean EEC for DCPA on turf (42 ug/liter) + Annual Mean EEC for TPA on turf (321 ug/liter).
D. The shallow ground water combined EEC is 275 ug/liter for turf and 192 ug/liter for cotton

5.3 Chronic Aggregate Risk

Since no chronic residential handler or post application scenarios have been identified, chronic DWLOCs for DCPA were calculated based on anticipated residues in food alone. These are presented in Table 16 and indicate that DWLOCs for are less than the combined EECs for both surface water and shallow ground water. It should be noted that the EECs for the metabolite TPA are much higher than the EECs for DCPA and are the risk drivers.

Table 16 - DCPA Summary of Chronic DWLOC Calculations for DCPA Applied to Turf

Population Subgroup	cPAD (mg/kg/day)	Food Exposure (mg/kg/day)	Available Water Exposure[A] (mg/kg/day)	DWLOC[B] (ug/liter)	Surface Water Combined EEC for Turf[C] (ug/liter)	Surface Water Combined EEC for Cotton[D] (ug/liter)
U.S. Population	0.01	0.000097	0.0099	347	363	377
Females 13-50 yrs	0.01	0.000088	0.0099	297	363	377
Children 1-6 yr	0.01	0.000113	0.0099	99	363	377
All Infants	0.01	0.000085	0.0099	99	363	377

A. Available Water Exposure = cPAD - food exposure
B. DWLOC = (Available water exposure X body weight X 1000 ug/mg)/liters of water
C. Surface Water Combined EEC for Turf = Annual Mean EEC for DCPA on turf (42 ug/liter) + Annual Mean EEC for TPA on turf (321 ug/l).
D. Surface Water Combined EEC for Cotton = Annual Mean EEC for DCPA on cotton (11 ug/l) + Annual Mean EEC for TPA on cotton (366 ug/l).

Note. The shallow ground water combined EEC is 275 ug/liter for turf and 192 ug/liter for cotton

5.4 Cancer Aggregate Risk Assessment

DCPA

Cancer DWLOCs were calculated using food alone and together with residential exposure data. The handler exposure scenario which resulted in the greatest risk (Scenario #1, Hand or Shaker Can Application to Garden Vegetables) was used in the calculation. DWLOC values were calculated for adults only and the results are shown in Tables 17 and 18. All of the cancer DWLOCs are less than the EECs, therefore the aggregate cancer risks may be of concern when drinking water is included, particularly when the metabolite TPA is combined with DCPA. The EECs for the metabolite TPA are much higher than the EECs for DCPA and are the risk drivers. The aggregate cancer risks excluding drinking water ranged from $1.5x10^{-8}$ when food alone is considered alone to 6.8×10^{-7} when food and residential handler exposures are considered.

Table 17 - Cancer DWLOC Calculations for DCPA (Using EECs Following Application to Turf)								
Exposure Scenario	Dietary Food Exposure (ug/kg/day)	Residential Exposure (ug/kg/day)	Aggregate Cancer Exposure (ug/kg/day)	Target Maximum Exposure[1] (ug/kg/day)	Max Water Exposure[2] (ug/kg/day)	Surface Water EEC[3] (ug/liter)	Ground Water EEC[4] (ug/liter)	Cancer DWLOC[5] (ug/liter)
Food Alone	0.097	N/A	0.097	2.0	1.9	193	275	67
Food and Home Gardener Handler (Hand Application)	0.097	0.35	0.45	2.0	1.6	193	275	54
Food and Home Lawn Post Application	0.097	0.23	0.33	2.0	1.7	193	275	59

1 Target Maximum Exposure (ug/kg/day) = $3.0 \times 10^{-6} / Q_1^*$ X 1000 ug/mg where Q1* = 1.5×10^{-3} mg/kg/day

2 Maximum Water Exposure (ug/kg/day) = [Target Maximum Exposure - (Food Exposure + Residential Exposure)]

3. Surface Water EEC = 36 Year Mean EEC for DCPA on turf (33 ug/liter) + 36 Year Mean EEC for TPA on turf (160 ug/liter).

4. Ground Water EEC = 36 Year Mean EEC for DCPA on turf (0.0088 ug/liter) +36 Year Mean EEC for TPA on turf (275 ug/liter).

5 Cancer DWLOC(µg/liter) = $\frac{\text{[maximum water exposure (ug/kg/day) x body weight (kg)]}}{\text{[water consumption (liter)]}}$

Table 18 - Cancer DWLOC Calculations for DCPA (Using EECs Following Application to Cotton)								
Exposure Scenario	Dietary Food Exposure (ug/kg/day)	Residential Exposure (ug/kg/day)	Aggregate Cancer Exposure (ug/kg/day)	Target Maximum Exposure[1] (ug/kg/day)	Max Water Exposure[2] (ug/kg/day)	Surface Water EEC[3] (ug/liter)	Ground Water EEC[4] (ug/liter)	Cancer DWLOC[5] (ug/liter)
Food Alone	0.097	N/A	0.097	2.0	1.9	228	192	67
Food and Home Gardener Handler (Hand Application)	0.097	0.35	0.45	2.0	1.6	228	192	54
Food and Home Lawn Post Application	0.097	0.23	0.33	2.0	1.7	228	192	59

1. Same as above for Table 17
2. Same as above for Table 17
3. Surface Water EEC = 36 Year Mean EEC for DCPA on cotton (8 ug/liter) + 36 Year Mean EEC for TPA on cotton (220 ug/liter).
4. Ground Water EEC = 36 Year Mean EEC for DCPA on cotton (0.014 ug/liter) +36 Year Mean EEC for TPA on cotton (192 ug/liter).
5. Same as above for Table 17

HCB

The aggregate cancer risks were calculated for HCB using food alone and together with residential exposure data. The HCB food exposures include HCB from DCPA as well as HCB from other pesticides as discussed in "Assessment of the Dietary Cancer Risk of Hexachlorobenzene and Pentachlorobenzene as impurities in Chlorothalonil, PCNB, Picloram, and several other pesticides". (DP Barcode D243499 of 2/26/98). These values are shown in Table 19.

Table 19 - Aggregate Cancer Risks for HCB				
Exposure Scenario	Dietary Food Exposure (ug/kg/day)	Residential Exposure (ug/kg/day)	Aggregate Cancer Exposure (ug/kg/day)	Aggregate Cancer Risk[2]
Food Alone[1]	6.3e-04	N/A	6.3e-04	6.5e-07
Food and Home Gardener Handler (Hand Application)	6.3e-04	1.7e-05	6.5e-04	6.6e-07
Food and Home Lawn Post Application (Irrigated)	6.3e-04	1.1e-05	6.4e-04	6.6e-07
Food and Home Lawn Post Application (Not Irrigated)	6.3e-04	3.0e-05	6.6e-04	6.8e-07

1. Includes HCB exposures from DCPA, chlorothalonil, PCNB, Picloram, endsulfan, chloropyrifos-methyl, atrazine, simazine and clopyrilid.
2. Aggregate Cancer Exposure (ug/kg/day) * 0.001 mg/ug X Q_1* where Q_1* = 1.0 mg/kg/day

EFED was not asked to estimate the amount of HCB in drinking water due to solely to current or proposed pesticide uses because of such an estimation is not possible due to environmental contamination from previous HCB uses and industrial waste streams. It is estimated, however, that HCB would partition primarily to the sediment because HCB has high soil sorption coefficient of 50,000 and a low water solubility of 0.005 mg/liter. Limited water data as reported in the ATSDR Toxicological Profile for HCB indicated that the average HCB level in the drinking water from Lake Ontario was 0.0001 ug/liter in 1982. The EPA Office of Water has established a maximum contaminant limit (MCL) of 0.001 mg/liter and a maximum contaminant level guideline (MCLG) of zero mg/liter for HCB.

The HCB cancer risks due to fish consumption were not calculated because significant levels of HCB are expected primarily in freshwater fish consumed by subsistence and sports fisherman. Also, it is not possible to determine what percentage of the HCB contamination is due to pesticidal sources. It should be noted that the consumption of contaminated fish which are not in interstate commerce (such as those caught by subsistence and sports fisherman) is regulated by state monitoring of chemical residues (including HCB) in fish and issuance of consumption advisories or bans by the EPA's Office of Water when hazardous residue levels are detected. Most of the advisories (96%) have been issued for elevated residue levels of five major pollutants (PCBs, dioxins, mercury, cadmium and chlordane) while the remaining advisories (4%) have been issued for a variety of other chemicals to include heavy metals, organochlorine pesticides, creosote, polyaromatic hydrocarbons and HCB. A fact sheet that describes the National Listing of Fish and Wildlife Advisories can be obtained from the EPA Office of Water at http://www.epa.gov/waterscience/fish/.

6.0 CUMULATIVE RISK

The Food Quality Protection Act (1996) stipulates that when determining the safety of a pesticide chemical, EPA shall base its assessment of the risk posed by the chemical on, among other things, available information concerning the cumulative effects to human health that may result from dietary, residential, or other non-occupational exposure to other substances that have a common mechanism of toxicity. The reason for consideration of other substances is due to the possibility that low-level exposures to multiple chemical substances that cause a common toxic effect by a common mechanism could lead to the same adverse health effect as would a higher level of exposure to any of the other substances individually. A person exposed to a pesticide at a level that is considered safe may in fact experience harm if that person is also exposed to other substances that cause a common toxic effect by a mechanism common with that of the subject pesticide, even if the individual exposure levels to the other substances are also considered safe.

For the purposes of this risk assessment, DCPA is being considered toxicologically unique. HED did not perform a cumulative risk assessment as part of this tolerance action because HED has not yet initiated a review to determine if there are any other chemical substances that have a mechanism of toxicity common with that of DCPA. For purposes of this tolerance action, EPA has assumed that DCPA does not have a common mechanism of toxicity with other substances.

Before undertaking a cumulative risk assessment, HED will follow procedures for identifying chemicals that have a common mechanism of toxicity as set forth in the "*Guidance for Identifying Pesticide Chemicals and Other Substances that Have a Common Mechanism of Toxicity*" (64 FR 5795-5796, February 5, 1999). On this basis, the petitioner must submit, upon EPA's request and according to a schedule determined by the Agency, such information as the Agency directs to be submitted in order to evaluate issues related to whether DCPA shares a common mechanism of toxicity with any other substance and, if so, whether any tolerances for DCPA need to be modified or revoked. If HED identifies other substances that share a common mechanism of toxicity with DCPA, HED will perform aggregate exposure assessments on each chemical, and will begin to conduct a cumulative risk assessment once the final guidance HED will use for conducting cumulative risk assessments is available.

HED has recently developed a framework that it proposes to use for conducting cumulative risk assessments on substances that have a common mechanism of toxicity. This guidance was issued for public comment on June 30, 2000 (65 FR 40644-40650) and is available from the OPP Website at: http://www.epa.gov/fedrgstr/EPA-PEST/2000/June/Day-30/6049.pdf. In the draft guidance, it is stated that a cumulative risk assessment of substances that cause a common toxic effect by a common mechanism will not be conducted until an aggregate exposure assessment of each substance has been completed. The proposed guidance on cumulative risk assessment of pesticide chemicals that have a common mechanism of toxicity is expected to be finalized by the summer of 2002.

7.0 OCCUPATIONAL EXPOSURE

An occupational exposure and risk assessment is required for an active ingredient if: (1) certain toxicological criteria are triggered and (2) there is potential exposure to handlers (i.e., mixers, loaders, applicators, etc.) during use. DCPA meets both criteria. DCPA is in acute toxicity category III by the acute dermal and inhalation exposure routes. There is potential exposure to occupational handlers during agricultural site applications of DCPA.

7.1 Occupational Handlers/Applicators

Workers may experience short term exposures to DCPA during mixing, loading and application. It is highly unlikely that applications would occur for more than 30 consecutive days, thus intermediate term or chronic exposures were not evaluated. Dermal exposures were not assessed for non-cancer risks because dermal toxicity testing indicated that the no effects were observed at the highest dose tested (1000 mg/kg/day). Dermal exposures were assessed for cancer risks using the dermal absorption factors of 22% for DCPA and 27% for HCB. HCB exposures were calculated assuming that DCPA contains 40 ppm HCB by weight as a manufacturing impurity. The non-cancer risks were calculated for inhalation exposures using the maximum proposed label rate, a standard ExpoSAC value for daily acres treated and protection factors for PPE specified on the Dacthal W-75 label. The MOE calculations are detailed in Appendix B and the results are summarized in Table 20. The MOE are greater than 100 when label required PPE is used and are not of concern.

Table 20 - Inhalation MOEs for DCPA Occupational Exposure and Non-Cancer Risks

Exposure Scenario	Crops	Application Rate (lb ai/Acre)	Treated Area (Acres/ day)	MOE with Baseline PPE	MOE with Label required PPE
1A - Open Mixing and Loading of Wettable Powder (PHED Data)	basil, celeriac, chicory, chives, coriander, dill, ginseng, marjoram, oriental radish, radicchio and parsley	10.5	80	96	480
1A - Open Mixing and Loading of Wettable Powder (Study Data)				350	1700
1B - Spray Application - Groundboom Open Cab				5600	28000

Baseline PPE includes no respirator
Label required PPE includes a dust/mist respirator for inhalation exposures (a protection factor of 5 was assumed)

The cancer risks were assessed for DCPA and HCB assuming one exposure day per year for application to minor crop fields. This assumption is conservative because one application will be made per season and the typical minor crop field is much less than 80 acres. Parsley and ginseng are grown on 5000 acres each while the other minor crops are grown on a total of 6000 acres. The cancer risk calculations are detailed in Appendix B and the results are summarized below in Tables 21 and 22.

Table 21- Occupational Handler Cancer Risks for DCPA					
Exposure Scenario	**Crops**	**Application Rate (lb ai/Acre)**	**Treated Area (Acres/day)**	**DCPA Cancer Risk With Baseline PPE**	**DCPA Cancer Risk With Label Required PPE**
1A - Open Mixing and Loading of Wettable Powder - PHED Data	Same Minor Crops as in Table 20	10.5	80	2.1×10^{-5}	1.1×10^{-6}
1A - Open Mixing and Loading of Wettable Powder - Study Data				No Data	2.6×10^{-7}
1B - Spray Application Using Open Cab Groundboom				9.4×10^{-8}	8.0×10^{-8}
Baseline PPE includes long pants, long sleeve shirt, no gloves and no respirator. Label Required PPE includes chemical resistant gloves, long pants, long sleeved shirt, hat and a dust/mist respirator .					

Table 22- Occupational Handler Cancer Risks for HCB in DCPA					
Exposure Scenario	**Crops**	**Application Rate (lb ai/Acre)**	**Treated Area (Acres/day)**	**DCPA Cancer Risk With Baseline PPE**	**HCB Cancer Risk With Label Required PPE**
1A - Open Mixing and Loading of Wettable Powder - PHED Data	Same Minor Crops as in Table 20	10.5	80	6.9×10^{-7}	3.6×10^{-8}
1A - Open Mixing and Loading of Wettable Powder - Study Data				No Data	8.3×10^{-9}
1B - Spray Application Using Open Cab Groundboom				3.0×10^{-9}	2.6×10^{-9}

It should be noted that the cancer risks were calculated only for the applications made to the minor crops and do not include the risks for the other crops such as onions and broccoli that are treated with DCPA. Vegetable growers typically grow a variety of vegetables and it is highly conceivable that onions, broccoli and parsley could be grown on the same farm. According to the 1997 Census of Agriculture, the acreage for onions and broccoli is 165,000 and 145,000 respectively**.** The risks of applying DCPA to vegetables was evaluated in the 1998 RED and was found to be acceptable **only if** label required single layer PPE was used. The risks were in the 10^{-5} range assuming 80 acres treated per day and ten days exposure per year. These assumptions are valid because the average and 94th percentile vegetable farm size is 70 and 250 acres according to the 1997 Census of Agriculture.

7. 2 Post-Application Exposures

No occupational post application exposures are anticipated because DCPA will be applied to minor crop fields before the seeds germinate. The Dacthal product label has a 12-hour restricted entry interval (REI). This REI complies with the Agency's Worker Protection Standard.

7.3 Incident Report

There were no incidents reported for DCPA in California during the years 1996 to 2000. The usage of DCPA declined from a maximum of 522,000 pounds in 1996 to 133,000 pounds in 2000 because DCPA was not produced from 1997 to 2000.

8.0 Data Gaps and Information Requirements

Although the DCPA databases were substantially complete, confidence in several areas of the risk assessment would improve with more data. The following data gaps were identified.

- A guideline 28-day inhalation study in rats, which must include assessment of thyroid histopathology and thyroid hormone levels, is required.

- A confirmatory study showing the comparative short-term thyroid toxicity of DCPA in adults and offspring is required. This study should include evaluation of thyroid hormone levels and liver induction. The registrant should consult with the Agency to discuss this study before beginning the study.

- The following additional data were required in the 1998 RED for confirmatory purposes: ruminant feeding study; poultry metabolism study; additional ILV testing and radiovalidation of the livestock method; storage time and temperature of samples from several older field trial studies; and large-scale field rotational crop studies on representative crops to which the registrant wishes to permit rotation. These data are not required for this assessment of new uses, however, because the existing and proposed uses of DCPA do not include animal feed crops.

The following information could be used to refine the risk estimates.

- Actual use rates for DCPA applied to residential turf and golf courses. The highest rate of 15 lbs ai/acre was used by EFED in the drinking water assessment. This rate is only applied in the fall for annual blue grass control. It is not known if the typical homeowner would attempt to control annual bluegrass since it is similar in appearance to desirable grass species. It is more likely that the homeowner would focus on crabgrass control which uses a lower rate of 10.5 lb ai/acre. Golf course managers would probably be interested in controlling both crabgrass and annual bluegrass, however, their use of DCPA is limited by the fact that DCPA cannot be used on putting greens.

- Actual use rates and percent cropped treated for cotton. The QUA report indicated that there was no reported use of DCPA on cotton for the years 1995-2000. NASS data for 2001 indicated that there was no reported usage of DCPA on the cotton crop in 2001.

Appendix A - DCPA Subchronic/Chronic Toxicity Profile

DCPA Subchronic/Chronic Tox Profile		
Study Type - Dose Levels	**NOAEL (mg/kg/day)**	**LOAEL (mg/kg/day)**
28-Day Feeding Rat (1990) Males: 0, 215, 860, or 1720 mg/kg/day Females: 0, 228, 890, or 1760 mg/kg/day MRID 41790901	<250	250 mg/kg/day based on hepatic hypertrophy. At 1720 mg/kg/day thyroid follicular cell hyperplasia in males. Thyroid hormones **not** evaluated.
90-Day Feeding Rat (1991) 0, 10, 50, 100, 150, or 1000 mg/kg/day MRID 41767901	50	100 based on centrilobular hypertrophy. At 1000 mg/kg/day there were gross and microscopic lesions of lungs and kidneys; microscopic lesions in thyroids; and increased liver weights. Thyroid hormones not evaluated.
2-yr Feeding/Carcinogenic Rat (1993) 0, 1, 10, 50, 500, 1000 mg/kg/day MRID42731001	1	10 based upon decreased T4 hormone and thyroid and liver histological changes. Increases in thyroid follicular cell adenomas and carcinomas, hepatocellular adenomas and carcinomas, and hepatocholangiomas in females.
Q1* = 1.5×10^{-3} based upon the three combined types of liver tumors in female rats (3/4 scaling factor)		
13-Week Feeding Mouse (1985) Males: 0, 625, 1250, 2500, or 7500 ppm , = 0, 100, 199, 406, or 1235 mg/kg/day Females: 0, 1000, 2500, 5000, 10000 ppm, = 0, 223, 517, 1049, or 2198 mg/kg/day MRID 41064801	Male: 406 Female: 1049	Males: 1235 Females: 2198 based on centrilobular hepatocyte enlargement. Thyroid hormones and thyroid histology not evaluated.
Carcinogenic Mouse (1988) Males: 0, 12, 123, 435, 930 mg/kg/day Females: 0, 15, 150, 510, 1141 mg/kg/day	510	1141 based upon elevated liver enzymes and increased liver wt in females. Increases in hepatic adenomas (females) and carcinomas (males, females).
13-Week Feeding Dog	Not available.	
1-yr Feeding Dog	Not available.	
Developmental Tox Rat (1986) 0, 500, 1000, 2000 mg/kg/day MRID 00160685	Maternal: > 2000 Develop: > 2000	Maternal: > 2000 Develop: > 2000
Developmental Tox Rabbit (1987 and 1989) 0, 500, 1000, 1500 mg/kg/day 0, 125, 250, 500 mg/kg/day MRID 41054820 (2 studies)	Maternal: 250 Develop: >500	Maternal: 500 based upon maternal mortality Develop: >500
2-Gen Reproduction Rat (1990) 0, 1000, 5000, or 20000 ppm = 0, 50, 250, or 1000 mg/kg/day F2b generation: 0, 200, 500, or 20000 ppm = 0, 10, 25, or 1000 mg/kg/day MRID 41750103	Parental: 50 Repro: >1000 Offspring: 50	Parental: 250 based upon body weight decrements, gross and microscopic changes in kidneys and lungs, and microscopic changes in liver and thyroids. Repro: >1000 Offspring: 250 based upon pup body weight decrements during the lactation period.
21-Day Dermal Toxicity in Rats (1989) 0, 100, 300, or 1000 mg/kg/day MRID 41231803	>1000	>1000 Thyroid hormones and histology not evaluated.
Subchronic Inhalation Study	Not available.	

DCPA Subchronic/Chronic Tox Profile		
Study Type - Dose Levels	**NOAEL (mg/kg/day)**	**LOAEL (mg/kg/day)**
Metabolism in rats: In 6 separate metabolism studies, ^{14}C-DCPA was given as single or multiple oral gavage doses to rats at 1 or 1000 mg/kg/day. There were no significant sex differences in any of the studies. Absorption was rapid and essentially complete by 48 hours. Absorption was more efficient at 1 mg/kg/day (79%-86% of administered dose) than at 1000 mg/kg/day (6-9%). Urine was the major route of excretion. Less than 1% of radiolabel was found in bile, so compound in feces represents unabsorbed compound. The major compound found in urine was the mono-methyl metabolite, 4-carbomethoxy-2,3,5,6-tetrachlorobenzoic acid. The di-acid metabolite, TPA, represented approximately 1% of radioactivity in urine. No DCPA was found in urine. Radiolabel did not bioaccumulate in tissues following repeated treatment. Although a high percentage of the administered dose was found in fat 12 hours after discontinuance of dosing (12% of dose in low-dose animals), radiolabel had rapidly depleted by 168 hours (0.03%). Concentration of radiolabel in the thyroid increased at 36 hours postdosing when compared to the 12 hour time period, however, radiolabel in the thyroid rapidly depleted by 168 hours. By 168 hours, highest concentration of radiolabel in both dose groups was in the kidney.		
Mutagenicity		
Mouse lymphoma assay	Negative for forward mutations	
Cytogenetic assay in CHO cells	Negative for clastogenicity	
UDS assay	Negative	
SCE in CHO cells	Negative	
Ames	classified unacceptable	
Dermal Absorption	22% including compound on skin at 47.5 ug/cm^2	

APPENDIX B

DCPA OCCUPATIONAL AND RESIDENTIAL HANDLER RISK ASSESSMENT TABLES

Table B1: Unit Exposure Data for DCPA Occupational and Residential Exposure Assessment				
Scenario	Mitigation Level[1]	Data Source	Unit Exposure Values (Per lb Ai Handled)	Data Confidence
Occupational Handler Scenarios				
1A - Mix/Load Wettable Powder (Open Mixing)	Baseline	PHED	Dermal = 3.7 mg Inhalation = 43.4 ug	Hand and dermal data are ABC grades. Hand = 7 replicates; Dermal = 22 to 45 replicates. Low confidence due to low number of hand replicates. Inhalation = 44 replicates with grade ABC data and medium confidence.
1A - Mix/Load Wettable Powder (Open Mixing)	Single Layer	PHED	Dermal = 0.17 mg Inhalation = 8.7 ug	Hand and dermal data are ABC grades. Hand replicates = 24 replicates; Dermal = 22 to 45 replicates. Medium confidence in hand and dermal data. Baseline inhalation data was used with a 80% protection factor to account for the use of PF5 dust masks.
1A - Mix/Load Wettable Powder (Open Mixing)	Baseline	MRID 435493-02	Dermal = N/A Inhalation = 12 ug	N =15 inhalation replicates with grade AB data. Medium confidence because not all of the replicates included a complete work cycle.
1A - Mix/Load Wettable Powder (Open Mixing)	Single Layer	MRID 435493-02	Dermal = 0.0375 mg Inhalation = 2.4 ug	N =15 hand, dermal and inhalation replicates with grade AB data. Medium confidence because not all of the replicates included a complete work cycle. Inhalation data are used with a 80% protection factor to account for PF5 dust masks.
1B - Groundboom Spray Application	Baseline	PHED	Dermal = 0.014 mg Inhalation = 0.74 ug	Hand and dermal data are AB grade. Hand = 29 replicates; Dermal = 23 to 42 replicates. High confidence in hand and dermal data. Inhalation data are AB grade with 22 replicates and high confidence.
1B - Groundboom Spray Application	Single Layer	PHED	Dermal = 0.014 mg Inhalation = 0.15 ug	The same dermal data are used as for baseline. Gloved hand data are ABC grades, with 21 replicates, and medium confidence level. The same inhalation data are used as for baseline and respirator use is not assumed.
Residential Handler Scenarios				
1 - Hand Application of Granules	None	PHED	Dermal = 114 mg Inhalation = 467 ug	N = 16 dermal ,hand and inhalation replicates with grade ABC data. Hand data was for gloved hand and required 10X adjustment for use without gloves.
2 - Belly Grinder Application	None	PHED	Dermal = 110 mg Inhalation = 62 ug	N = 20 to 45 dermal replicates, ABC grades. Hand replicates = 23, ABC grades. Medium Confidence. N = 40 Inhalation replicates, AB grades, High Confidence.
3. Load/Apply Granules with a Broadcast Spreader	None	ORETF[2]	Dermal = 0.68 mg Inhalation = 0.91 ug	Grade AB Data. N = 30 replicates. High Confidence despite large variability in results.

Notes for Table B1

1 Single Layer - chemical resistant gloves, long pants, long sleeved shirt, hat and a PF5 filtering face piece respirator (ie dustmask).

2. This study involved the application of granular Dacthal to residential lawns. It was reviewed by Health Canada and Gary Bangs in Document #D261948 and found to be acceptable.

Table B2: DCPA Inhalation MOEs for Occupational Handlers Using Baseline PPE

Exposure Scenario	Crops	Application Rates (lb ai/Acre)[a]	Treated Area (Acres/day)[b]	Inhalation Daily Exposure (mg/day)[c]	Inhalation Absorbed Daily Dose (mg/kg/day)[d]	Inhalation MOE[e]
1A - Mix/Load Wettable Powder (PHED Data)	Low Acreage Row Crops Including Parsley	10.5	80	36	0.52	96
1A - Mix/Load Wettable Powder (Study Data)				10	0.14	347
1B - Spray Application - Groundboom				0.62	0.0089	5631

Table B3: DCPA Inhalation MOEs for Occupational Handlers Using Label Required PPE
(Label Requires Dust Mist Respirator)

Exposure Scenario	Crops	Application Rates (lb ai/Acre)[a]	Treated Area (Acres/day)[b]	Inhalation Daily Exposure (mg/day)[c]	Inhalation Absorbed Daily Dose (mg/kg/day)[d]	Inhalation MOE[e]
1A - Mix/Load Wettable Powder (PHED Data)	Low Acreage Row Crops Including Parsley	10.5	80	7.22	0.1032	484
1A - Mix/Load Wettable Powder (Study Data)				2.02	0.0288	1736
1B - Spray Application - Groundboom				0.13	0.0018	27778

Notes

a Maximum label rate

b Taken from Exposac Policy #9 “Standard Values for Daily Acres Treated in Agriculture”

c Daily Exposure (mg/day) = Application Rate (lb ai/Acre) * Treated Area (Acre/day) * Unit Exposure Value (µg exposure/ lb ai handled) *[1mg/1000µg (conversion factor)].

d Absorbed Daily Dose (mg/kg/day) = Daily Exposure (mg/day) * Absorption Factor (1.0 for inhalation) ÷ Body Weight (70kg).

e MOE (unitless) = NOAEL (mg/kg/day) ÷ Combined Absorbed Daily Dose (mg/kg/day). Where NOAEL = 50 mg/kg/day for short or intermediate -term exposures. of 100 or greater is acceptable for DCPA short or intermediate term exposures.

A Margin of Exposure (MOE)

Table B4: DCPA Occupational Handler Cancer Risks Using Baseline PPE (1 exposure day per Year)

Exposure Scenario	Crops	Application Rates (lb ai/Acre)	Treated Areas (Acres/day)	Daily Exposure (mg/day)[a]		Absorbed Daily Dose (mg/kg/day)[b]		Combined Lifetime Absorbed Daily Dose (mg/kg/day)[c]	DCPA Cancer Risk[d]
				Dermal	Inhalation	Dermal	Inhalation		
1A - Mix/Load Wettable Powder - PHED Data	Low Acreage Row Crops Including Parsley	10.5	80	3108	36	9.77	0.52	1.4e-02	2.1e-05
1A - Mix/Load Wettable Powder - Study Data				N/a	N/A	N/A	N/A	N/A	N/A
1B - Spray Application - Groundboom				11.8	0.62	0.037	0.0089	6.3e-05	9.4e-08

Table B5: DCPA Occupational Handler Cancer Risks Using Label Required PPE (1 exposure day per Year)

Exposure Scenario	Crops	Application Rates (lb ai/Acre)	Treated Areas (Acres/day)	Daily Exposure (mg/day)[a]		Absorbed Daily Dose (mg/kg/day)[b]		Combined Lifetime Absorbed Daily Dose (mg/kg/day)[c]	DCPA Cancer Risk[d]
				Dermal	Inhalation	Dermal	Inhalation		
1A - Mix/Load Wettable Powder - PHED Data	Low Acreage Row Crops Including Parsley	10.5	80	142.8	7.22	0.449	0.1032	7.6e-04	1.1e-06
1A - Mix/Load Wettable Powder - Study Data				31.5	2.02	0.099	0.0288	1.8e-04	2.6e-07
1B - Spray Application - Groundboom				11.8	0.13	0.037	0.0018	5.3e-05	8.0e-08

Notes

a Daily Exposure (mg/day) = Application Rate (lb ai/Acre) * Treated Area (Acre/day) * Unit Exposure Value (mg or µg exposure/ lb ai handled) *[1mg/1000µg (conversion factor if necessary)].

b Absorbed Daily Dose (mg/kg/day) = Daily Exposure (mg/day) * Absorption Factor (0.22 for dermal; 1.0 for inhalation) ÷ Body Weight (70kg).

c Combined Lifetime Averaged Daily Dose (mg/kg/day) = Combined Potential Daily Dose (see note below) * 1 Annual Treatment Days / 365 days per year * 35 years working / 70 year lifespan.
Note - Combined Potential Daily Dose (mg/kg/day) = Dermal Potential Daily Dose (mg/kg/day) + Inhalation Potential Daily Dose (mg/kg/day).

d Carcinogenic Risk = Combined Lifetime Averaged Daily Dose (mg/kg/day) * Q_1^* $(mg/kg/day)^{-1}$. Q_1^* = 0.0015 for DCPA.

Table B6: HCB Occupational Handler Cancer Risks Using Baseline PPE (1 day per Year)

Exposure Scenario	Crops	Application Rates (lb ai/Acre)	Treated Areas (Acres/day)	HCB Daily Exposure (mg/day)[a]		Absorbed Daily Dose (mg/kg/day)[b]		Combined Lifetime Absorbed Daily Dose (mg/kg/day)[c]	HCB Cancer Risk[d]
				Dermal	Inhalation	Dermal	Inhalation		
1A - Mix/Load Wettable Powder - PHED data	Low Acreage Row Crops Including Parsley	10.5	80	1.2e-01	1.5e-03	4.7e-04	2.1e-05	6.7e-07	6.9e-07
1A - Mix/Load Wettable Powder - Study Data				N/a	N/A	N/A	N/A	N/A	N/A
1B - Spray Application - Groundboom				4.7e-04	2.5e-05	1.8e-06	3.6e-07	2.9e-09	3.0e-09

Table B7: HCB Occupational Handler Cancer Risks Using Label Required PPE (1 day per Year)

Exposure Scenario	Crops	Application Rates (lb ai/Acre)	Treated Areas (Acres/day)	HCB Daily Exposure (mg/day)[a]		Absorbed Daily Dose (mg/kg/day)[b]		Combined Lifetime Absorbed Daily Dose (mg/kg/day)[c]	HCB Cancer Risk[d]
				Dermal	Inhalation	Dermal	Inhalation		
1A - Mix/Load Wettable Powder - PHED data	Low Acreage Row Crops Including Parsley	10.5	80	5.7e-03	2.9e-04	2.2e-05	4.2e-06	3.5e-08	3.6e-08
1A - Mix/Load Wettable Powder - Study Data				1.3e-03	8.1e-05	4.8e-06	1.2e-06	8.1e-09	8.3e-09
1B - Spray Application - Groundboom				4.7e-04	5.0e-06	1.8e-06	7.2e-08	2.5e-09	2.6e-09

Notes

a Daily Exposure (mg/day) = DCPA Application Rate (lb ai/Acre) * Acres Treated/Day * Unit Exposure Value * 0.00004 HCB contamination factor in DCPA

b Absorbed Daily Dose (mg/kg/day) = Daily Exposure (mg/day) * Absorption Factor (0.265 for dermal; 1.0 for inhalation) ÷ Body Weight (70kg).

c Combined Lifetime Averaged Daily Dose (mg/kg/day) = Combined Potential Daily Dose (see note below) * 1 Annual Treatment Day / 365 days per year * 50 years working / 70 year lifespan.
Note - Combined Potential Daily Dose (mg/kg/day) = Dermal Potential Daily Dose (mg/kg/day) + Inhalation Potential Daily Dose (mg/kg/day).

d Carcinogenic Risk = Combined Lifetime Averaged Daily Dose (mg/kg/day) * Q_1^* $(mg/kg/day)^{-1}$. Q_1^* = 1.0 for HCB

Table B8 - DCPA Inhalation MOES for Homeowner Applications (No PPE is Used)

Exposure Scenario	Crops	Application Rate (lb ai/Acre)[a]	Treated Area (Acre/day)[b]	Daily Inhalation Exposure (mg/day)[c]	Absorbed Daily Dose (mg/kg/day)[d]	DCPA MOE[e]
1 - Apply Granules by Hand or Shaker Can	Garden Vegetables	10.5	0.023	1.1e-01	1.6e-03	31034
2 - Load/Apply Granules with a Belly Grinder	Garden Vegetables	10.5	0.023	1.5e-02	2.1e-04	233754
3A - Load/Apply Granules with a Broadcast Spreader	Garden Vegetables	10.5	0.500	4.8e-04	6.8e-06	7326007
3B - Load/Apply Granules with a Broadcast Spreader	Lawns	15.0	0.500	6.8e-04	9.8e-06	5128205

Notes

a Maximum Label Rates
b. Treated Area = Based upon Revised SOP Assumption of 2/2001 (1000 SF = 0.023 acre)
c Daily Inhalation Exposure (mg/day) = Application Rate (lb ai/Acre) * Treated Area (Acre/day) * Unit Exposure Value (µg exposure/ lb ai handled) *[1mg/1000µg (conversion factor)].
d Absorbed Daily Dose (mg/kg/day) = Daily Exposure (mg/day) * Absorption Factor (1.0 for inhalation) ÷ Body Weight (70kg).
e DCPA MOE = 50/Absorbed Daily Dose where 50 = NOAEL (mg/kg/day) for short or intermediate term inhalation exposures.
An MOE of 100 or greater is acceptable for short or intermediate term exposures.

Table B9 - DCPA Cancer Risk for Homeowner Applications (Two Treatment Days per Year)

Exposure Scenario	Crops	Application Rates (lb ai/Acre)	Treated Areas (Acre/day)	Daily Exposure (mg/day)[a]		Absorbed Daily Dose (mg/kg/day)[b]		Combined Lifetime Absorbed Daily Dose (mg/kg/day)[c]	DCPA Cancer Risk[d]
				Dermal	Inhalation	Dermal	Inhalation		
1 - Apply Granules by Hand or Shaker Can	Garden Vegetables	10.5	0.023	28	1.1e-01	8.7e-02	1.6e-03	3.5e-04	5.2e-07
2- Load/Apply Granules with a Belly Grinder	Garden Vegetables	10.5	0.023	27	1.5e-02	8.3e-02	2.1e-04	3.3e-04	4.9e-07
3A - Load/Apply Granules with a Broadcast Spreader	Garden Vegetables	10.5	0.500	3.6	4.8e-04	1.1e-02	6.8e-06	4.4e-05	6.6e-08
3B - Load/Apply Granules with a Broadcast Spreader	Lawns	12.5	0.500	4.3	5.7e-04	1.3e-02	8.1e-06	5.2e-05	7.8e-08

a Daily Exposure (mg/day) = Application Rate (lb ai/Acre) * Treated Area (Acre/day) * Unit Exposure Value (mg or µg exposure/ lb ai handled) *[1mg/1000µg (conversion factor if necessary)].
b Absorbed Daily Dose (mg/kg/day) = Daily Exposure (mg/day) * Absorption Factor (0.22 for dermal; 1.0 for inhalation) ÷ Body Weight (70kg).
c Combined Lifetime Averaged Daily Dose (mg/kg/day) = Combined Potential Daily Dose (see note below) * 2 Annual Treatment Days / 365 days per year * 50 years exposure / 70 year lifespan.
Note - Combined Potential Daily Dose (mg/kg/day) = Dermal Potential Daily Dose (mg/kg/day) + Inhalation Potential Daily Dose (mg/kg/day).

d Carcinogenic Risk = Combined Lifetime Averaged Daily Dose (mg/kg/day) * Q_1^* $(mg/kg/day)^{-1}$. Q_1^* = 0.0015 for DCPA.

Table B10 - HCB Cancer Risk for Homeowner DCPA Applications (Two Treatment Days per Year)

Exposure Scenario	Crops	Application Rates (lb ai/Acre)	Treated Areas (Acre/day)	Daily Exposure (mg/day)[a]		Absorbed Daily Dose (mg/kg/day)[b]		Combined Lifetime Absorbed Daily Dose (mg/kg/day)[c]	HCB Cancer Risk[d]
				Dermal	Inhalation	Dermal	Inhalation		
1 - Apply Granules by Hand or Shaker Can	Garden Vegetables	10.5	0.023	1.1e-03	4.5e-06	4.2e-06	6.4e-08	1.7e-08	1.7e-08
2- Load/Apply Granules with a Belly Grinder	Garden Vegetables	10.5	0.023	1.1e-03	6.0e-07	4.1e-06	8.6e-09	1.6e-08	1.6e-08
3A - Load/Apply Granules with a Broadcast Spreader	Garden Vegetables	10.5	0.500	1.4e-04	1.9e-08	5.5e-07	2.7e-10	2.2e-09	2.2e-09
3B - Load/Apply Granules with a Broadcast Spreader	Lawns	12.5	0.500	1.7e-04	2.3e-08	6.6e-07	3.3e-10	2.6e-09	2.6e-09

a Daily Exposure (mg/day) = Same as above for DCPA with a 0.00004 HCB contaminant factor to account for the fact that DCPA contains 40 PPM HCB as a manufacturing impurity.

b Absorbed Daily Dose (mg/kg/day) = Daily Exposure (mg/day) * Absorption Factor (0.27 for dermal; 1.0 for inhalation) ÷ Body Weight (70kg).

c Combined Lifetime Averaged Daily Dose (mg/kg/day) = Combined Potential Daily Dose (see note below) * 2 Annual Treatment Days / 365 days per year * 50 years exposure / 70 year lifespan.
Note - Combined Potential Daily Dose (mg/kg/day) = Dermal Potential Daily Dose (mg/kg/day) + Inhalation Potential Daily Dose (mg/kg/day).

d Carcinogenic Risk = Combined Lifetime Averaged Daily Dose (mg/kg/day) * Q_i^* $(mg/kg/day)^{-1}$. $Q_i^* = 1.0$ for HCB

APPENDIX C

DCPA RESIDENTIAL
POST APPLICATIONEXPOSURE AND
RISK ASSESSMENT TABLES

Table C1: Input Values for Residential Post Application Turf Risk Assessment

Average Label Application Rate (lb ai/acre):		12.5
Maximum Label Application Rate (lb ai/acre):		15.00
Study Application Rate (lb ai/acre):		21.00
Limit of Quantification (ug/cm^2):		0.004
Transferable Residue (% of Rate) For Object-to-Mouth Ingestion Exposure Assessment		20
Transferable Residue (% of Rate) For Hand-to-Mouth Ingestion Exposure Assessment		5
Transferable Residue (% of Study Rate) from TTR Study:		1.78
TTR Data Source:		416488-6
Slope of Semilog Regression for HAT 2 to DAT 14 on Irrigated Turf		-0.0631
DAT 0 Initial DCPA TTR from Study prior to irrigation (ug/cm^2):		4.2
DAT 0 Initial DCPA TTR from Study following irrigation (ug/cm^2)		1.6
Adult Dermal Exposure Duration On Lawns (hr/day):		2
Toddler Dermal Exposure Duration On Lawns (hr/day):		2
Toddler Hand-to-Mouth Duration On Lawns (hr/day):	2	
Adult Dermal Exposure Duration While Golfing (hr/day):		4
Long-term Adult Dermal Transfer Coefficient (TC) On Lawns (cm^2/hr):	7300	
Long-term Adult Dermal TC While Golfing (cm^2/hr):		250
Toddler Hand Surface Area (cm^2/both hands):		20
Toddler Short-Term Frequency of Hand-to-Mouth Events (events/hour):		20
Object-to-Mouth Surface Area Contacted (cm^2 mouthed):		25
Soil Ingestion (mg soil ingested/day):		100
Soil Density (cm^3/gram):		0.67
Saliva Extraction Factor :		0.50
Typical Activity Homeowner (days/yr):		4
Typical Activity Golfer (days/yr):		4
Activity Duration (yrs):		50
Lifetime (yr):		70
Days/yr:		365
Level of Concern for MOEs		100
NOAEL (mg/kg/day) for Dermal Exposures (any duration):		N/A
Oral NOAEL (mg/kg/day) for Incidental Oral Exposure (any duration):		50
Adult Body Weight (kg):		70
Toddler Body Weight (kg):		15
DCPA Q1* (mg/kg/day)-1:		0.0015
DCPA Dermal Absorption Factor (DA):		0.22
HCB Q1* (mg/kg/day)-1:		1.0
HCB Dermal Absorption Factor (DA):		0.27

Table C2: Irrigated Turf and Soil Residue Levels

DAT	TTR for Adult Dermal[1] (ug/cm^2)	TTR for Hand to Mouth[2] (ug/cm^2)	TTR for Object to Mouth[3] (ug/cm^2)	[Soil] For Ingestion[4] (ppm)
0	0.952	8.42	33.7	112.8
1	0.894			
2	0.839			
3	0.788			
4	0.740			
5	0.695			
6	0.652			
7	0.612			
8	0.575			
9	0.540			
10	0.507			
11	0.476			
12	0.447			
13	0.419			
14	0.394			
Average	0.635			

Notes

1. TTR for Adult Dermal = 12.5 lb ai/acre /21.0 lb ai/acre * 1.6 ug/cm^2 x $e^{DAT \times -0.0631}$

2. TTR for Hand-to-mouth = 15 lb ai/acre x 0.05 x (4.54×10^8 µg/lb) x (2.47×10^{-8} $acre/cm^2$)

3. TTR for Object-to-mouth = 15 lb ai/acre x 0.20 x (4.54×10^8 µg/lb) x (2.47×10^{-8} $acre/cm^2$)

4. Soil Residue for Ingestion = 15 lb ai/acre x 1.0 x (4.54×10^8 µg/lb) x (2.47×10^{-8} $acre/cm^2$) x 0.67 cm^3/gram

Table C3A: Adult Cancer Risks for DCPA Applied to Irrigated Turf

DAT	**TTR**	**Dermal Exposure On Residential Turf**			**Dermal Exposure While Playing Golf**		
	ug/cm^2	LADD[1] (mg/kg/day)	Cancer Risk per Annual Day of Exposure	# of Annual Exposure Days at 1.0e-06	LADD (mg/kg/day)	Cancer Risk per Annual Day of Exposure	# of Annual Exposure Days at 1.0 x 10-6
AVG	0.64	5.7E-05	8.6e-08	11.7	3.9e-06	5.9e-09	171

AVG = Average of DAT 0 to DAT 14.

1. LADD = TTR x TC x 0.001 mg/µg x DA x hours exposure/day x (1/body weight) x 1/365 x 50/70

2. Cancer Risk per Annual Day of Exposure = LADD x Q_1* where Q_1* = 0.0015 for DCPA

3. # of Annual Exposure Days at 1.0×10^{-6} = 1.0×10^{-6}/(Cancer Risk per Day of Exposure)

Table C3B: Adult Cancer Risks for HCB from DCPA Applied to Irrigated Turf

DAT	TTR	Dermal Exposure On Residential Turf			Dermal Exposure While Playing Golf		
	ug/cm^2 (Note 1)	$LADD^2$ (mg/kg/day)	Cancer Risk per Annual Day of Exposure[3]	# of Annual Exposure Days[4] at 1.0×10^{-6}	$LADD^2$ (mg/kg/day)	Cancer Risk per Annual Day of Exposure[3]	# of Annual Exposure Days[4] at 1.0×10^{-6}
AVG	0.00003	2.8E-09	2.9e-09	350	1.9e-10	2.0e-10	>365

Notes

1. TTR = DCPA TTR (ug/cm^2) X HCB contamination factor (0.0004 or 40 PPM).
2. LADD = TTR x TC x DA x hours exposure/day x (1/body weight in kg) x 1/365 x 50/70
3. Cancer Risk per Annual Day of Exposure = LADD x Q_1* where Q_1* = 1.0 for HCB
4. # of Annual Exposure Days at 1.0×10^{-6} = 1.0×10^{-6}/(Cancer Risk per Day of Exposure)

Table C4: Incidental Oral Non-Cancer Risks for Exposure to Turf

DAT	TTR for HTM[1]	Hand to Mouth Risk		TTR for OTM[1]	Object to Mouth Risk		[Soil] for Ingestion[1]	Soil Ingestion Risk		Aggregate MOE[6]
	(ug/cm^2)	Dose[2]	MOE[5]	(ug/cm^2)	Dose[3]	MOE[5]	(ppm)	Dose[4]	MOE[5]	
0	8.42	0.224	220	33.7	0.056	890	113	7.5e-04	66500	180

1. See Table C2
2. Hand to Mouth Dose (mg/kg/day) = (TTR x 0.50 x 20 cm^2/event x 20 events/hr x 10^{-3} mg/µg x 2 hours/day)/15 kg
   Note: 0.50 = saliva extraction factor
3. Object to Mouth Dose (mg/kg/day) = (TTR x 25 cm^2/day x 10^{-3} mg/µg)/15 kg
4. Soil Ingestion Dose (mg/kg/day) = (100 mg soil/day x 10^{-6} g/µg)/15 kg
5. MOE = NOAEL/Dose
6. Aggregate MOE = NOAEL / (Hand to Mouth Dose + Object to Mouth Dose + Soil Ingestion Dose)

Table C5: Turf and Soil Residue Levels for Non-Irrigated Turf

DAT	TTR for Dermal (ug/cm^2)
0	2.50[1]
1	2.35[2]
2	2.20
3	2.07
4	1.94
5	1.82
6	1.71
7	1.61
8	1.51
9	1.42
10	1.33
11	1.25
12	1.17
13	1.10
14	1.03
Average	1.67

Notes

1. TTR for Dermal on DAT 0 = 12.5 lb ai/acre /20.1 lb ai/acre * 4.2 ug/cm^2

2. TTR after DAT 0 = Previous Day's TTR - 10 percent

Table C6A: Adult Cancer Risks for DCPA on Non-Irrigated Turf

DAT	**TTR**	**Dermal Exposure On Residential Turf**			**Dermal Exposure While Playing Golf**		
	ug/cm^2	LADD[1] (mg/kg/day)	Cancer Risk per Annual Day of Exposure[2]	# of Annual Exposure Days[3] at 1.0 x 10-6	LADD[1] (mg/kg/day)	Cancer Risk per Annual Day of Exposure[2]	# of Annual Exposure Days[3] at 1.0 x 10-6
AVG	1.67	1.5E-04	2.2e-07	4.5	1.0e-05	1.5e-08	65

AVG = Average of DAT 0 to DAT 14

1. LADD = TTR x TC x 0.001 mg/µg x DA x hours per day exposure x (1/body weight) x 1/365 x 50/70

2. Cancer Risk per Annual Day of Exposure = LADD x Q_1* where Q_1* = 0.0015 for DCPA

3. # of Annual Exposure Days at 1.0 x 10^{-6} = 1.0 x 10^{-6}/(Cancer Risk per Day of Exposure)

Table C6B: Adult Cancer Risks Values for HCB on Non-Irrigated Turf

DAT	TTR	Dermal Exposure On Residential Turf			Dermal Exposure While Playing Golf		
	ug/cm^2 (Note 1)	$LADD^2$ (mg/kg/day)	Cancer Risk per Annual Day of Exposure[3]	# of Annual Exposure Days[4] at 1.0 x 10-6	$LADD^2$ (mg/kg/day)	Cancer Risk per Annual Day of Exposure[3]	# of Annual Exposure Days[4] at 1.0 x 10-6
AVG	6.7e-05	7.4e-09	7.5e-09	133	5.0e-10	5.1e-10	>365

1. TTR = DCPA TTR (ug/cm^2) X HCB contamination factor (0.0004 or 40 PPM).

2. LADD = TTR x TC x 0.001 mg/µg x DA x hours exposure/day x (1/body weight in kg) x 1/365 x 50/70

3. Cancer Risk per Annual Day of Exposure = LADD x Q_1* where Q_1* = 1.0 for HCB

4. # of Annual Exposure Days at 1.0 x 10^{-6} = 1.0 x 10^{-6}/(Cancer Risk per Day of Exposure)